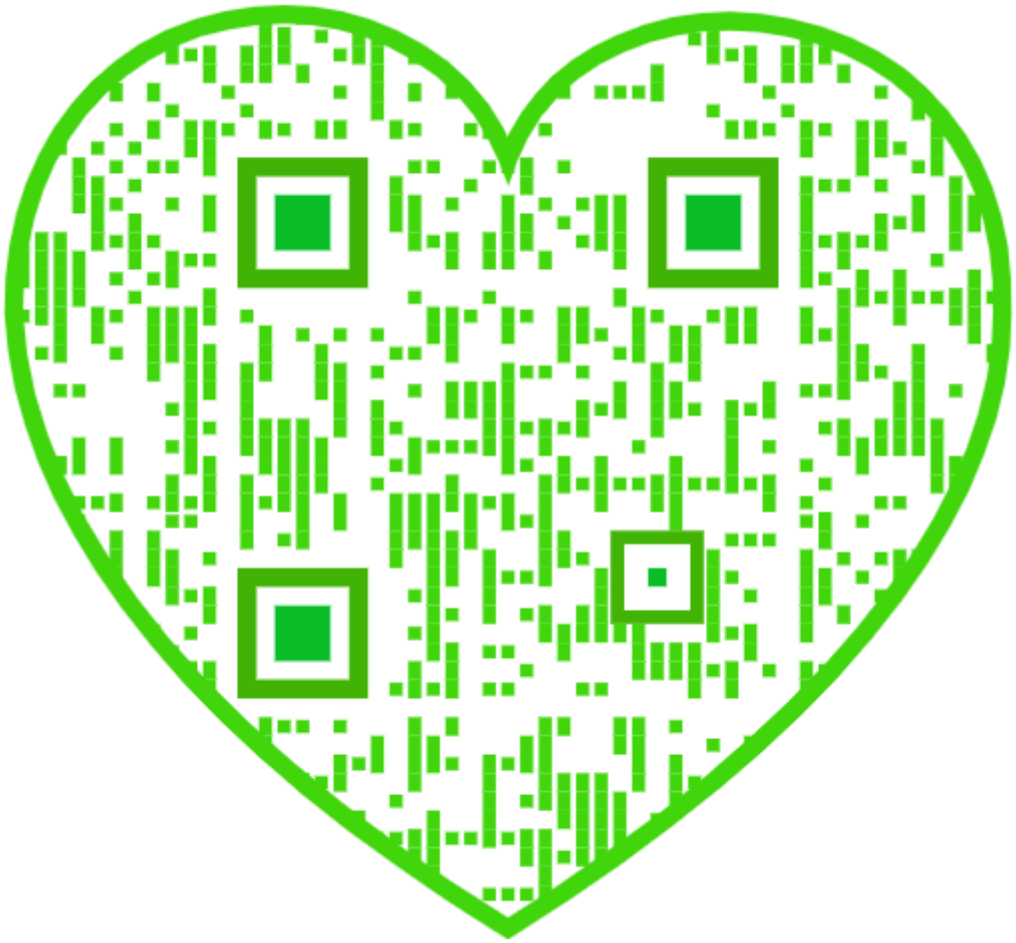

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INTERNATIONALLY DANGEROUS FOREST TREE DISEASES

MISCELLANEOUS PUBLICATION NO. 939
FOREST SERVICE
U.S. DEPARTMENT OF AGRICULTURE

Internationally Dangerous Forest Tree Diseases

By Working Group on International Cooperation in Forest Disease Research, Section 24, Forest Protection, International Union of Forestry Research Organizations

Supplement to Section 24–14, Proceedings of the 13th Congress, International Union of Forestry Research Organizations, Vienna 1961

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Forest Service

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FOREWORD

One of the hazards of international trade is the possible movement of dangerous insects and pathogens accompanying the exchange of agricultural and forest products. To illustrate, of the major plant pests and diseases that cause losses in excess of a billion dollars annually and necessitate costly control programs in the United States, over half were introduced from other continents. Included among the major introduced diseases are three that have taken and continue to take a tremendous toll from North American forests: chestnut blight, white pine blister rust, and Dutch elm disease. All practical precautions should be taken to prevent the introduction of additional dangerous forest pathogens and every effort made to provide advance information on eradication or control procedures for those that may be accidentally introduced.

The accomplishment of these objectives is dependent on international cooperation to recognize and characterize those forest tree pathogens in each continent considered to be potentially dangerous if introduced to other continents and to promote measures to restrict their spread. To that end, the Forest Service of the United States Department of Agriculture has participated in the work of the International Union of Forestry Research Organizations, particularly in the activities of the Working Group on International Cooperation in Forest Disease Research. The report of this Working Group to the 13th Congress of the parent organization in Vienna in 1961 is a significant contribution to improved forest disease prevention and control in all lands. The Forest Service, therefore, welcomes the opportunity to publish it for worldwide distribution and use.

This publication is truly the result of cooperative effort. It assembles under one cover the reports of 37 scientists from 14 countries in Asia, Europe, and North America. It includes descriptions of 57 forest pathogens in the Northern Hemisphere considered to be potentially most damaging to forest production in other continents, with recommendations for special precautions to restrict their intercontinental spread. These diseases occur on 87 genera of forest trees, of which 50 genera are important in the United States.

The text of the separate reports is with few exceptions as submitted by the various authors. No attempt has been made to adopt a uniform system of nomenclature for pathogens and hosts.

This publication is a Supplement to Section 24–14 of the Proceedings, 13th Congress, International Union of Forestry Research Organizations, Vienna, 1961.

V. L. Harper
Deputy Chief, Forest Service
U.S. Department of Agriculture
(Vice President, IUFRO)

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PROGRESS REPORT: WORKING GROUP ON INTERNATIONAL COOPERATION IN FOREST DISEASE RESEARCH

Organization and Objectives

J. R. Hansbrough, Chairman

Director, Division of Forest Disease Research, Forest Service, U.S. Department of Agriculture, Washington 25, D.C., U.S.A.

One of the topics considered at the 12th IUFRO Congress, Oxford, 1956, was the problem of the intercontinental spread of forest tree diseases and the need for international cooperation to lessen their threat and to reduce losses from those already introduced abroad. Examples were cited of the catastrophic losses in the past from introduced forest pathogens such as *Endothia parasitica* (Murr.) P. J. & H. W. And., *Cronartium ribicola* J. C. Fisch., and *Ceratocystis ulmi* (Buis.) Moreau, respectively the causes of chestnut blight, white pine blister rust, and the wilt disease of elm. There was complete agreement on the need for increased international cooperation in forest disease research.

Four measures were recommended for immediate attention: (1) Literature reviews of the forest diseases of all continents, (2) studies by pathologists of one continent of the forest diseases of other continents, (3) test plantings of all species in a genus to determine their susceptibility to a specific disease (e.g., oak species susceptibility to oak wilt), and (4) test plantings of the most important tree species of each continent on other continents to determine their susceptibility to native diseases.

In 1957 a working group in Section 24 was organized to carry out these recommendations. Membership included representatives from Asia (India and Japan), Europe (Austria, England, Italy, Netherlands, Norway, and Yugoslavia), and North America (Canada and the United States). The objectives of this working group were to secure more information on the forest tree diseases of all continents, to evaluate their potential impact on important forest tree species of other continents, and to develop and improve methods of preventing the spread of diseases from one continent to another.

It was agreed that initially the working group would concentrate its activities primarily on the diseases of three genera—*Pinus*, *Populus*, and *Quercus*—in the North Temperate Zone. Each member would attempt to stimulate special studies of the diseases of these genera in his country and adjacent countries and see that the results were published. In addition, special efforts would be made by all to insure distribution of new information on important forest diseases to pathologists and foresters in other countries and continents.

We meet here today to review our accomplishments as individuals and as a group. We have carried on research or stimulated others to do so on forest disease problems in our respective areas. We have

released and distributed publications. We have made some progress in establishing plantations of exotic forest trees to determine their susceptibility to local diseases. Above all, but much more difficult to appraise and document, we have made an excellent start in creating a public awareness of the magnitude of forest disease losses and in promoting serious consideration of methods of reducing them.

One evidence of the effectiveness of our work to date is this symposium in which we will review selected dangerous forest diseases in the continents of the North Temperate Zone. If the information thereby made available is used to deter or prevent the international spread of even one of the pathogens discussed, this Congress will be a success and our working group will have justified its establishment.

Our efforts and our progress so far are but the first step toward our ultimate objective of lessening forest disease losses in all continents. We will discuss the possibilities of broadening our group activities to stimulate forest disease research, education, and action programs throughout the world. One way would be to organize another symposium in the near future with worldwide coverage of selected pathogens considered to be most dangerous if introduced to other continents. Other ways would be (1) to encourage preparation and wide distribution of reports listing under each genus of important forest trees the name, diagnostic characteristics, and distribution of dangerous pathogens; (2) to recommend more effective quarantine procedures; and (3) to organize a corps of specialists in each continent responsible for promoting forest disease research, prevention, and control activities.

As chairman of your working group, I want to express my sincere appreciation to each member for your hearty cooperation in our activities, especially for the way that you have participated in the preparations for this Congress. Also, I wish to thank our section leader, Dr. Biraghi, and our president, Mr. Macdonald, for their invaluable assistance in scheduling our meetings and otherwise contributing to our objectives. With the continued enthusiastic support of all concerned, I am sure that our future contributions to the advancement of forest disease control programs can be increasingly significant.

ACCOMPLISHMENTS: ASIA

Japan

ROKUYA IMAZEKI

Director, Division of Forest Protection, Government Forest Experiment Station, Meguro, Tokyo, Japan

Many kinds of exotic trees, introduced from continental Asia, Europe, North America, and other countries, have been planted in Japan during the past 70 or more years. Some of them were cultivated in arboreta for merely botanical purposes, but others were planted in various parts of the country on a larger scale for silvicultural experiments. These experimental plantations, however, were prompted simply for experiment's sake or sometimes for curiosity and, therefore, no systematized investigations have been carried out.

After the war, the loss of forests in overseas colonies, the drain of resources, and the greatly increased timber demand led to extreme disorganization in timber supply and demand, and consequently the policy of increasing timber production became the urgent problem to be solved in Japanese forestry. Among ways of solving this problem, the introduction of exotic trees is considered promising and an organized survey of existing plantations of exotic trees is considered essential.

At that time, the proposal on the cooperative international investigation of the diseases of exotic trees was presented to Prof. N. Takahasi, the former representative of the Working Group of Eastern Asia, by Dr. A. Biraghi and Dr. J. R. Hansbrough.

Though Prof. Takahashi did not specialize in forest pathology, he had a profound comprehension of the importance of the forest disease and pest problem as applying to future forestry in Japan. He visited the Government Forest Experiment Station in Toyko in order to deliberate this proposal with me and Dr. K. Ito, the chief of the Section of Forest Pathology in our Division. Needless to say, there could be no two opinions as to the advisability and urgency of the matter, and we decided willingly to accept this proposal, and to organize the Working Group of Eastern Asia.

We recommended Prof. Takahashi as the representative of the Working Group for the time being, and decided that the other two should actively push forward the work.

The first project undertaken by the Working Group was to carry out a fundamental survey of the diseases and pests of exotic trees which had been introduced into Japan to that time.

In order to accomplish this as quickly as possible, it was very necessary to ask for the help of plant pathologists and entomologists in the agricultural field. Accordingly, we consulted with Prof. N. Hiratsuka and others, who are mentioned below, and succeeded in organizing a joint research group. The joint research was financially supported by the Forestry Agency and started in the spring of 1959, working to a 3-year program. The members of this group are as follows:

Person in charge.—Prof. N. Hiratsuka, Tokyo University of Education.

Pathologists.—Prof. N. Hiratsuka, ex-Prof. Senji Kamei, Hokkaido University; Prof. Nobukiyo Takahashi, Tokyo University; Asst. Prof. Katsuya Nakamura, Tokyo University of Agriculture and Technology; Prof. Shigeyasu Akai, Kyoto University; Prof. Iwao Hino, Yamaguchi University; Dr. Yoshikazu Nishikado, Director, the Nishikado Mycological Institute, formerly Prof. of Okayama University.

Entomologists.—Prof. Toichi Uchida, Hokkaido University; Prof. Masatoshi Nittoh, Tokyo University; Dr. Yoshihiko Kurosawa, Curator, National Science Museum; Prof. Teiichi Okutani, Hyogo Agricultural College; Asst. Prof. Magoshiro Moritsu, Yamaguchi University.

At the start of this research, we focused our attention on the following kinds of trees as the main objects for the investigation: *Acacia dealbata* and *A. mollissima*, *Eucalyptus robusta* and *E. globulus*, *Populus* spp., *Larix europaea* and *L. dahurica*, *Picea excelsa*, and *Pinus* spp. (*banksiana*, *elliottii*, *koraiensis*, *palustris*, *radiata*, *rigida*, *strobus*, *sylvestris*, and *taeda*).

The researchers have made steady progress during the past 2 years. Recently, Prof. N. Hiratsuka presented an intermediate report of the results obtained in 1959 to the Forestry Agency. According to this report, the research members collected 475 specimens of diseases and 355 specimens of injurious insects from various regions. The identifications are not yet completed.

While this joint research was being carried on, the forest pathologists and entomologists in the Government Forest Experiment Station were also working on the same studies in cooperation with the joint research group. Annually a meeting has been held to discuss the results obtained and to facilitate further studies on them.

From these investigations, it became clear that we must pay more careful attention to the following diseases.

Poplar diseases. Dieback of poplars caused by *Diaporthe medusae* Nit. prevails in Honsyu, the middle part of Japan. Dieback and canker caused by *Cytospora chrysosperma* and *Leucostoma nivea* occurs in Hokkaido, the northern region of Japan. Among five kinds of rust diseases, leaf rust caused by *Melampsora larici-populina* Kleb. is the most common and serious. *Marssonia brunnea* (E. & Ev.) Magn. attacks leaves and young shoots. Poplars belonging to the sections Aigeros and Tacamahaca are severely damaged. *Septotis populiperda* (Moesz & Smarods) Wat. & Cash which causes the leaf blotch is believed to have been introduced with cuttings imported from abroad.

Acacia diseases. *Glomerella* (*Physalospora*) *acaciae* Ito & Shibukawa causes very severe damage to nursery stock.

Eucalyptus diseases. Damping-off caused by *Pythium*, *Fusarium*, *Rhizoctonia*, and *Cylindrocladium* are very severe.

Larch diseases. Shoot blight caused by *Physalospora laricina* Sawada is very dangerous in northern regions of Japan. I shall report further on this disease later on. *Larix europaea* and *L. dahurica* are very susceptible.

Pine diseases. *Pinus sylvestris* is very susceptible to *Cronartium quercuum*. Young plantations of *P. strobus* are sometimes attacked

by four kinds of needle rust very severely and also by *Armillaria* root rot. Nursery stocks of *P. radiata* are very heavily attacked by *Diplodia pineae*. *P. radiata*, *P. canariensis*, *P. nigra*, *P. luchuensis*, *P. strobus* are very susceptible to the leaf blight caused by *Cercospora densiflorae* Hori & Nambu.

Among many exotic pines, *Pinus strobus* is one of the most promising in Japan and is now planted everywhere in this country. The plantation area will be increased year by year. In this situation of plantation planting with this exotic, the forest pathologists must exercise utmost precaution against the occurrence of blister rust. Needless to say, we must establish whatever effective countermeasures are necessary to prevent the introduction of this disease from abroad and as quickly as possible.

While this work is still going on, it is considered very necessary to make a thorough survey in order to ascertain whether the fungus *Cronartium ribicola* is present or not on native *Ribes* spp. in Japan, although we have not been aware of the occurrence of the blister rust on Japanese native five-leaved pines.

Pathologists of the Government Forest Experiment Station, Hokkaido University, and Tokyo University searched for *C. ribicola* on wild and cultivated *Ribes* spp. throughout Hokkaido in 1959. During this cooperative survey they collected some *Cronartium* growing on some *Ribes* from several parts of Hokkaido. It is uncertain whether the collected *Cronartium* is exactly the same as the true *C. ribicola* although the fungus seems very similar to the latter morphologically. This important question remains to be solved quickly.

This is my report covering the past 3 years. I will take this opportunity to express our sincere gratitude to Prof. N. Takahashi who did so much to establish the foundation of our Working Group. He retired from the representative position of the Group in Japan and recommended me in place of him in June last year.

I shall make every effort to accomplish the significant objective of this international cooperative investigation on the forest disease problem, keeping close contact with the forest pathologists in Korea and China with whom I, to my regret, have not yet been able to get an opportunity to talk over the activity of the Working Group.

India

B. K. Bakshi

Head, Division of Forest Protection, Forest Research Institute, New Forest (Dehra Dun), India

Reports for India have been prepared on the parasitic fungi on *Pinus*, *Populus*, and *Quercus* and on the exotic forest trees that have been successfully established in India. Requests were sent to the following countries for information on the diseases of *Pinus*, *Populus*, and *Quercus:* Burma, Cambodia, Ceylon, Indonesia, Laos, Malaya, Pakistan, Philippines, Singapore, Thailand, and Vietnam.

Pakistan responded with a list of the parasitic fungi on *Pinus* and *Quercus;* Burma with a list of the species of *Pinus* occurring naturally in that country, with the notation that no information is available on diseases; and the Philippines with a brief report on the diseases of the two species of *Pinus* in that country. The remaining countries replied

that no pertinent information was available.

In this report on the parasitic fungi on *Pinus*, *Populus*, and *Quercus* in India, the diseases will be rated as follows: XXX-Dangerous, XX-May be dangerous, X-Not dangerous.

Pinus

Six species of *Pinus* occur in the forests of India: (1) *P. armandi* Frauchet, a Chinese pine, which occurs in northeastern parts of India, (2) *P. gerardiana* Wall. ex Lamb, localized in NW. Himalayas, (3) *P. insularis* Endl., in E. India, Upper Burma, and Philippines, (4) *P. merkusii* Jungh & De Vr., a tropical pine in Far East, (5) *P. roxburghii* Sargent, and (6) *P. wallichiana* Jack., the last two essentially Himalayan. Diseases are known for only three of the species. They are as follows:

1. *Pinus insularis* Endl. (Khasya pine). Eastern Distribution—Khasia and Naga hills, Manipur, Upper Burma, and Philippines.

Fomes pinicola Swartz. ex Fr. (XX)
Attacks standing trees, causes brown cubical rot.

Peridermium sp. (XX)
The infection results in large woody outgrowths or galls on stem. The alternate host of the rust is not yet known, but is believed to be *Quercus griffithi* on which III develops.

2. *Pinus roxburghii* Sargent (Chir pine). Distribution: Outer ranges of Himalayas, and on ridges of the Siwaliks flanking the Himalayas, Bhutan in the east to Afganistan in the west.

Capnodium pini Berk. and Curt. (X)

Coleosporium campanulae (Pers.) Lev. (X)
A heteroecious needle rust, 0 and I on needles of chir, II and III on *Campanula colorata* Wall.

Cronartium himalayense Bagchee (XXX)
A heteroecious stem rust, 0 and I on 3 needle chir pine, II and III in *Swertia* spp.

Fomes annosus (Fr.) Cke. (XX)

Fomes pini (Thore) Lloyd (XX)

Fomes pinicola Swartz. ex Fr. (XX)
Attacks standing trees, causes brown cubical rot.

Ganoderma applanatum (Pers.) Pat. (X)

Ganoderma lucidum (Leyss.) Karst. (X)

Lophodermium pinastri (Schrad.) Chev. (X)
Causes needle cast.

Pestalotia funerea Desm. (X)
On needles.

Polyporus schweinitzii Fr. (X)
Causes brown cubical rot in butt portions.

Septoria pisi Berk. (X)
Causes leaf spot.

3. *Pinus wallichiana* Jackson (blue pine). Distribution: Throughout temperate region of the Himalayas, chiefly at 6,000–

10,000 ft., sometimes ascending to 12,000 ft., and descending to 4,000 ft. Absent in Sikkim and many areas in Kumaon.

Armillaria mellea (Vahl.) Quel. (X)

Capnodium pini Berk. & Curt. (X)

Coleosporium barclayense Bagchee (XX)
A heteroecious rust, 0 and I on 5-needled blue pine, II and III on *Senecio* spp.

Cronartium ribicola Fischer (XX)
A heteroecious stem rust, 0 and I on 5-needled blue pine, II and III on Ribes.

Fomes annosus (Fr.) Cke. (XX)

Fomes fomentarius (L. ex Fr.) Kickx (X)
On dead wood.

Fomes pini (Thore) Lloyd (XXX)

Lophodermium pinastri (Schrad.) Chev. (X)
Causes needle cast.

Melampsora oblonga Bagchee (X)
Causes needle rust.

Polyporus schweinitzii Fr. (X)
On dead wood.

Polyporus tomentosus Fr. (X)
The fungus occasionally attacks blue pine. The parasitic form is referred to as *P. tomentosus* var. *circinatus*.

Populus

There are six species of poplars in India, either indigenous or introduced of which *Populus ciliata* Wall. and *P. euphratica* Oliv. are the most important. The others, like *P. nigra* Linn. and *P. alba* Linn., are planted for shade or ornament or are quite local.

1. *Populus alba* Linn. Distribution: Western Himalayan at 4,000–10,000 ft. extending west to Europe, in the Mediterranean and north to Siberia.

Melampsora rostrupii G. Wagner (X)
II and III on undersurface of leaves; I is not known in India.

2. *Populus ciliata* Wall. Distribution: Himalayan from Kashmir to Bhutan, at 4,000–10,000 ft.

Boerlagella effusa Syd. and Butler (X)
On wood and decorticated branches.

Cytospora chrysosperma (Pers.) Fr. (XX)
On branches. The *Cytospora* stage flourishes best during a wet summer. The *Valsa* stage may be found in plenty on the thicker branches in autumn.

Fomes fomentarius (L. ex Fr.) Kickx (X)

Linospora populina (Pers.) Schr. (X)
On leaves.

Melampsora ciliata Barc. (X)
II and III on undersurface of leaves; I is not

Nectria cinnabarina (Tode) Fr. (XX)
On branches.

Taphrina aurea (Pers.) Fr. (X)
On leaves.

Uncinula salicis (DC.) Wint. (XX)
On leaves.

3. *Populus euphratica* Olivier. Distribution: In Sind (Pakistan) along the Indus River, southern Punjab along the Sutlej River, Beluchistan, Ladakh, Tibet, ascends from plains up to 13,500 ft. Westwards it extends to Afganistan, Turkistan, Persia, Mesopotamia, and Palestine.

Cytospora sp. (Probably *C. chrysosperma*) (X)
On branches.

Quercus

Indian oaks occur in the Himalayas. Of a total of about 10 species in the eastern Himalayas, the most important are *Quercus lamellosa* Sm., *Q. pachyphylla* Kurz, *Q. lineata* Bl., and *Q. spicata* Sm. Of the 5 species in the western Himalayas, *Q. incana* Roxb., *Q. dilatata* Lindl., and *Q. semecarpifolia* Sm. are important.

1. *Quercus dealbata* Hook. Fils. & Th. Distribution: Bhutan, Khasi hills, Manipur.

Bispora catenula (Lev.) Sacc. (X)
On leaves.

2. *Quercus dilatata* Lindl. Distribution: Western Himalaya from Nepal westwards, chiefly at 7,000–9,000 ft.

Fomes fomentarius (L. ex Fr.) Kickx (XX)
Attacks living but more commonly dead standing and fallen logs. Causes white spongy rot with black zone lines.

Fomes rimosus Berk. (X)
A wound parasite; attacks both living and dead wood in which white stringy rot is produced.

Lenzites repanda (Mont.) Fr. (X)
On stems; rare.

Polyporus cuticularis (Bull.) Fr. (X)
On dead wood.

Polyporus obtusus Berk. (XX)
A wound parasite. Causes heart and sap rot (white spongy rot).

Pucciniopsis quercina Wakef. (X)
On leaves.

3. *Quercus griffithi* Hook. f. Distribution: Bhutan, Assam hills, hills of Burma east of Irrawaddy and Sittang.

Cronartium spp. (X)
III on leaves. 0 and I which occur on *Pinus insularis* may be the alternate stages.

4. *Quercus incana* Roxb. Distribution: Western Himalayas, extending eastward to Nepal, chiefly in outer ranges at 4,000–8,0000 ft.

Armillaria mellea (Vahl.) Quel. (X)

Bulgaria polymorpha (Oed.) Wett. (X)
Causes stem canker.

Diplodiella crustacea Karst. (X)
Causes dieback.

Fomes caryophylli (Rac.) Bres. (X)
Causes heart rot.

Fomes senex Nees & Mont. (XX)
A wound parasite on living and also on dead trees.
Pestalotia saccardoi Speg. (X)
Polyporus cuticularis (Bull.) Fr. (X)
On dead wood.

5. *Quercus lineata* Bl. Distribution: Eastern Himalayas, at 6,000–9,000 ft., Khasi and Naga hills (Assam), hills of Upper Burma.

Fomes pinicola Fr. (X)
Fomes senex Nees & Mont. (XX)
A wound parasite.

6. *Quercus semecarpifolia* Smith. Distribution: Throughout Himalayas, Assam-Burma frontier, China. A high altitude oak, usually at 8,000–12,000 ft.

Bulgaria inquinars Fr. (X)
Causes stem canker.
Fomes fomentarius (L. ex Fries) Kickx (XX)
On living trees and also on dead wood. Causes white spongy rot with zone lines.
Fomes rimosus Berk. (X)
A wound parasite on stems. Causes white stringy rot.
Polyporus consors (Berk.) Stev. (X)
On stems, causes white fibrous rot.
Polyporus cuticularis (Bull.) Fr. (X)
On butts of trees, fruiting on wound scars. Causes white stringy rot with chocolate-colored to black zone lines.
Polyporus gilvus Schwein. (X)
Polyporus obtusus Berk. (XX)
On living and dead trees. Usually a wound parasite. Causes white spongy rot.
Polyporus sulphureus Fr. (XX)
Causes brown cubical heart rot.
Polyporus versicolor (L.) Fr. (X)
Causes white spongy rot with light brown zone lines.
Stereum princeps Jungh. (XX)
A common wound parasite. Causes white pocket rot.

7. *Quercus sp.* Distribution: Not reported.

Lasiobotrys elegans (Syd.) Theiss. (X)
Leucoconis erysiphina Syd. (X)
On leaves.
Phyllosticta exigua Syd. (X)
On leaves.
Trichothyriella quercigena (Berk.) Theiss. (X)
On leaves.
Hydnum erinaceus Bull. (X)

Successful Exotics in India

Extract from "Exotics in Indian Forestry Practice" by S. K. Seth and S. N. Dabral. Proc. IX Silvic. Conf. (Part II), Dehra Dun, India, pp. 72–80. 1956.

Name of the species	*The states in which found successful*
Acacia abyssinica	Bihar (Palamau, Naterhat plateau).
A. decurrens	Madras and Uttar Pradesh hills.
A. melanoxylon	Madras (Aramby block).
A. mollissima	Assam, Himachal Pradesh, Madras (Nilgiris), and Uttar Pradesh (hills).
A. pycnantha	Uttar Pradesh and Madras hills.
Acer negundo	Jammu and Kashmir.
Albizzia falcata	Assam.
Aleurites fordii	Himachal Pradesh (Solan), Madras (Top Slip, Ootacamund and Wynaad Centres), Uttar Pradesh, lower hills and submountain areas.
A. montana	Himachal Pradesh (Solan hills), Uttar Pradesh in submountain regions.
Alnus incana	Bengal (Takdeh, Lingding, Raman).
Broussonetia papyrifera	Bengal (Bamanpokri), Bombay (Shahpur, Dandeli, Haliyal, Teregalli, Donga), Madras (Begur, Top Slip), Uttar Pradesh (moister areas in Bhabar and Dun).
Callitris rhomboidea	Madras (Nilambur, Aramby block).
Castanea sativa	Himachal Pradesh, Punjab (Manali).
Castelloa elastica	Bengal (Takdeh).
Casuarina jhunghuhniana	Madras (Chenglepat division).
C. montana	Madras (Nilgiris).
C. quadrivalvis	Madras (Carinhill blocks).
Catalpa japonica	Jammu and Kashmir.
Chamaecyparis lawsoniana	Bengal (Takdeh).
C. obtusa	Punjab (Manali).
Chlorophora excelsa	Madras in mixed deciduous forests, Assam, Sylhet division, Lawacherm plantation.
Cocos coronata	Bombay (Poona, Bhanubundra, Dona).
Cryptomeria japonica	Bengal (Pagalhora), Madras (Carinhill block), Punjab (Rahini, Kulu).
Cupressus arizonica	Jammu and Kashmir.
C. cashmiriana	Bengal (Takdeh).
C. horizontalis	Bihar (Palamau).
C. lusitanica	Bengal (Takdeh), Bihar (Palamau, Naterhat plateau).
C. macropoda	Bihar (Palamau, Naterhat plateau).
C. patula	Bengal (Takdeh).
C. torulosa	Bengal (Takdeh), Madras (Nilgiris), Punjab (Manali).
Enterolobium saman	Bengal (Bamanpokri).
Eucalyptus citriodora	Bombay (Londha, Haliyal, Dandeli).
E. globulus	Madras (Nilgiris).
E. maculata	Bombay (Shahpur).
E. tereticornis	Assam.
Fagus sylvatica	Punjab (Kulu, Manali).

Name of the species	*The states in which found successful*
Fraxinus excelsior	Jammu and Kashmir, Punjab (Kulu, Manali).
Hevea brasiliensis	Bengal (Sukna).
Juniperus virginiana	Bengal (Takdeh).
Khaya anthotheca	Bombay (Dandeli).
K. grandiflora	Bombay (Dandeli, Poona, Shahpur, Bhiltar, Lingmala).
K. senegalensis	Bombay (Dandeli, Shahpur, Poona, Binllu, Donga, Haliyal).
Kichia africana	Bengal (Sukna).
Larix decidua	Punjab (Kulu division, Manali, Pinetum, Nagani, Rahini).
L. griffithii	Punjab (Kulu, Manali, Rahini, etc.).
L. kaempheri	Punjab (Kulu, Manali, Rahini, etc.).
L. leptolepis	Punjab (Kulu, Manali, Rahini, etc.).
Laucaena glauca	Assam, Himachal Pradesh, Madras and Uttar Pradesh.
Ochroma lagopus	Madras; in all research centres of Madras, Wynaad, Southern Kanara, Palaghat, Nilambur, S. Coimbatore.
Pinus canariensis	Jammu and Kashmir.
P. caribaea	Assam.
P. halepensis	Jammu and Kashmir.
P. khasia	Bengal.
P. insignica	Madras (Nilambur).
P. laricio	Bengal (Raman), Punjab (Manali, Kulu).
P. macituna	Bihar (Palamau, Naterhat plateau).
P. massoniana	Bengal (Raman).
P. patula	Bengal (Raman), Bihar (Palamau, Naterhat plateau), Punjab (Kulu, Manali).
P. pinaster	Punjab (Kulu, Rahini, Manali).
P. radiata	Assam.
P. sylvestris	Punjab (Kulu, Manali, Pinetum).
P. taeda	Punjab (Kulu, Manali, Pinetum).
P. thunbergia	Bengal (Takdeh, Samunden).
Populus boroliensis	Uttar Pradesh (Chakrata 4500′).
P. gelerica	Uttar Pradesh (West Almora 6000′).
P. generosa	Punjab (Kulu, Manali, Pinetum).
P. monilifera	Punjab (Kulu, Manali, Pinetum).
P. robusta	Uttar Pradesh (Chakrata 4500′).
Prosopis juliflora	Bombay (Donja, Poona, Khandesh, Dandeli), Madhya Pradesh, (West Berar), Himachal Pradesh (between 2000–3000′), Madras (in dry fuel forests), PEPSU, Rajasthan (all through).
Pseudotsuga taxifolia	Punjab (Kulu, Manali, Rahini, Nagani, Nakas).
Quercus suber	Uttar Pradesh (6000–7000′), growth is very slow.

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Name of the species	*The states in which found successful*
Robinia pseudoacacia	Bihar (Naterhat plateau, Palamau), Jammu and Kashmir and Himachal Pradesh (6000–9000′).
Salix caerulea	Himachal Pradesh, Jammu and Kashmir.
Swietenia macrophylla	Bengal (the species is doing very well all over), Bombay (Dandeli).
S. mahogani	Bombay (Donga, Poona, Khandla, Dandeli).
Tilia europaea	Punjab (Manali, Pinetum).
Thuja gigantea	Bihar (Palamau).
T. orientalis	Bihar (Palamau).
T. plicata	Bengal (Raman), Punjab (Kulu, Manali, Rahini, Mahangarh, Dharmsala).

Pakistan

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In this report on the parasitic fungi on *Pinus* and *Quercus* in Pakistan, the diseases will be rated as follows: XXX—Dangerous, XX—May be dangerous, X—Not dangerous.

Pinus

The genus *Pinus* is represented in Pakistan by three species: (1) *P. roxburghii* Sargent, (2) *P. wallichiana* Jackson, and (3) *P. gerardiana* Wall. ex Lamb. The known diseases of the former two species are given below.

1. *Pinus roxburghii* Sargent (Chir pine)

Coleosporium campanulae (Pers.) Lev. (X)
A needle rust which may be serious at times, causes early shedding of needles.

Cronartium himalayense Bagchee (XX)
Rare due to dry situation where the pine grows.

Fomes annosus (Fr.) Cke. (XX)
Damage is not significant, except in very moist localities.

Fomes pini (Thore ex Fr.) Karst. (XX)
Rare.

2. *Pinus wallichiana* Jackson (blue pine)

Armillaria mellea (Vahl.) Quel. (XX)
Causes root rot with white stringy decay in wood.

Cenangium ferruginosum (Fr.) Fr. (XX)
Kills young plants, about 10 years old, in moist localities.

Coleosporium barclayense Bagchee (X)
Causes early shedding of needles.

Cronartium ribicola Fischer (XX)
The fungus is too rare to be of any consequence.

Dasyscypha fusco-sanguinea Rehm emend Hohnel. (XX)
Kills young plants.

Fomes annosus (Fr.) Cke. (XX)
Damage is not significant, except in very moist localities.
Fomes pini (Thore ex Fr.) Karst. (XXX)
A serious disease causing heart rot.
Fomes pinicola Swartz. ex Fr. (XX)
Attacks standing trees, causing brown cubical rot.
Leptostroma ahmadii Petr. (X)
On needles.
Lophodermium pini-excelsae Ahmad (X)
Causes needle cast in moist and shady localities.
Peniophora gigantea (Fr.) Mass. (X)
On dead wood. The fungus is a primary colonizer of dead stumps in preference to *Fomes annosus* (Fr.) Cke.
Polyporus gilvus (Schw.) Fr. (X)
On roots, rare.
Polyporus schweinitzii Fr. (XX)
Causes butt rot.
Polyporus versicolor (L.) Fr. (X)
Causes white spongy rot. (X)
Stereum purpureum Pers. (X)
Wound parasite. Causes white spongy rot.

Quercus

The genus *Quercus*, in Pakistan, is represented by 5 species: (1) *Q. dilatata* Lindl., (2) *Q. ilex* Linn, (3) *Q. incana* Roxb., (4) *Q. semecarpifolia* Smith, (5) *Q. gluca.* The diseases of the first four are given below.

1. *Quercus dilatata* Lindl.

Amphisphaeria fallax de Not. (X)
On bark.
Colpoma quercinum Wallr. (X)
Coryneum umbonatum Nees (X)
Causes girdling of twigs.
Dasyscypha indica (Cash) Ahmad (XX)
Causes canker on which fruit bodies appear.
Discosia atrocreas Tod. ex Fr. (X)
Causes leaf spot.
Fistulina hepatica Huds. ex Fr. (X)
Fomes fomentarius (L.) Fr. (XX)
Wound parasite. Causes white spongy rot.
Fomes pectinatus Klotz. (X)
Causes white pocket rot in oak.
Fomes rimosus Berk. (X)
Fomes torulosus Pers. ex Lloyd (X)
Causes heart rot.
Ganoderma applanatum (Pers.) Pat. (X)
Ganoderma lucidum (Leyss.) Karst. (XX)
Nummularia bulliardi Tul. (XX)
Causes dieback.
Penzigia quercum Mueller and Ahmad (X)
Polyporus cuticularis Bull. ex Fr. (X)
Causes heart rot.

Polyporus squamosus (Huds.) Fr. (X)
Causes brown cubical heart rot.
Stereum purpureum Pers. (X)
Wound parasite. Causes white spongy rot.
Trabutia quercina (Fr. & Rud.) Sacc. & Reun (X)
Causes leaf spot.
2. *Quercus ilex* Linn.
Amphisphaeria striata Niessl (X)
3. *Quercus incana* Roxb.
Fomes fastuosus Lev. (X)
Fomes pectinatus Klotz. (X)
Causes white pocket rot.
Fomes scruposus (Fr.) Cunn. (X)
Wound parasite, causing white spongy rot in heartwood.
Fomes semitosus Berk. (X)
Fomes senex Nees & Mont. (X)
Causes heart rot.
Ganoderma lucidum (Leyss.) Karst. (X)
Nummularia bulliardi Tul. (X)
Causes dieback.
Polyporus hispidus (Bull.) Fr. (X)
Causes heart rot.
Polyporus schweinitzii Fr. (X)
Causes brown cubical rot.
4. *Quercus semecarpifolia* Smith
Fomes senex Nees & Mont. (X)

Philippines

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Pinus

There are two indigenous species of pines in the Philippines; namely, *Pinus insularis* Endl. and *Pinus merkusii* Jungh. & Devr. The following diseases are known for these species:

1. *Pinus insularis* Endl. (Benguet pine)

The pure Benguet pine forest in the Philippines forms a distinct forest type (pine type) in the highlands of northwestern Luzon Island at elevations ranging from 500 to 2,500 meters above sea level but is best developed at elevations ranging from 900 to 1,500 meters. It is reported to exist also in Indo China, Burma, and India.

Fusarium sp. Dangerous
Causes damping-off.
Rhizoctonia sp. Dangerous
Causes damping-off.
Trametes sp. Unimportant
Causes wood rot.
Fomes sp. Unimportant
Causes wood rot.

Damping-off caused by *Rhizoctonia* spp. and *Fusarium* spp. is a common disease of nursery seedlings of Benguet pine in the Philippines. Two- to six-weeks-old seedlings are most susceptible to this disease. Effective control measures found by experiments are: 40% sulfuric acid (H_2SO_4) diluted in 1,000 cc. of water applied to every square foot of seedbed immediately after sowing the seeds; control of the density of sowing because too dense sowing favors the occurrence of the disease; formaldehyde (40% U.S.P.) diluted with water six times its volume used for treating the soil; and zinc oxide or red copper oxide as soil dressing, the former being more effective.

Fungi of the genera *Trametes* and *Fomes* have been observed to infect old and large trees over 60 cm. d.b.h.

2. *Pinus merkusii* Jungh. & Devr. (Mindoro pine or tapulau)

This species occurs in Zambales province and Mindoro Island in places as low as 100 meters altitude. It is reported to exist also in Burma, Indo China, Borneo, Thailand, and Sumatra.

There is no disease so far reported or observed affecting this species.

Burma

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Pinus

The following species of *Pinus* occur naturally in the forests of Burma:

Pinus insularis Endl. (*P. khasya* Royle.) The Khasi pine. Distribution: Hills of Upper Burma, i.e., the Chin Hills, the Pakokku Hills and between the Sittang and Salween Rivers and the Shan States. Chipwi valley, Hpimaw fort and Ngawchang valley in the Kachin State 2,500 feet a.s.l. upwards.

Pinus merkusii Jungh. The Tenasserim pine. Distribution: Southern Shan States southward through the hills of the Salween and Thaungyin drainages. 500 to 2,500 ft. a.s.l.

P. armandi Franchet. Armand's pine. Distribution: Panwa and Hpimaw pass, Kachin State.

P. wallichiana A. B. Jacks (*P. excelsa* Wall.) The Blue Pine. Distribution: Lukpyi in Kachin State.

Exotic species of *Pinus* growing in the Botanical Gardens, Maymyo are:

P. roxburghii Sarg. (*P. longifolia* Roxb.) The chir pine.
P. caribaea Morelet. The Cuban pine.
P. halepensis Miller. The Aleppo pine.
P. halepensis Miller var. *Orutia* (Tenore) Elwes & Henry. The Calabrian pine. } Postwar

None of these have so far been successfully raised in plantations.

No research appears to have been done on the diseases of pine. While at Maymyo I have noticed cases of witches'-broom on *P. insularis*.

Quercus

No research appears to have been done on the diseases of *Quercus* of which there are at least 40 species occurring in Burma.

ACCOMPLISHMENTS: EUROPE

Yugoslavia

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My principal activity since the creation of the Working Group has been surveying and checking important forest pathogens on *Pinus*, *Quercus*, and *Populus* in some of the countries of southern and eastern Europe. Because of the variety of forest conditions and the different research organizations in these parts of Europe, it has not yet been possible to cover the entire area as originally proposed. Personal contacts have been established with Dr. Biraghi, Chairman of Section 24, IUFRO, and later with Dr. Lohwag (Austria), Mr. Leontovyc (Czechoslovakia), and Dr. Kailidis (Greece). During this 3-year period, I have checked pathogens in Czechoslovakia, Greece, Italy, and Yugoslavia; the other forestry ranges will be checked during the next period of time. A completed list of the diseases present in southern and eastern Europe can be expected later, as a cooperative work, I believe, with Dr. Lohwag.

As a result of this work to date, several pathogens can be designated as specific for this part of Europe and as dangerous to other countries. Some additional organisms are also more or less common in northern and western Europe. The selected pathogens on *Pinus*, *Quercus*, and *Populus* in southern Europe are the following:

Pinus

Cronartium asclepiadeum (Willd.) Fr., agent of cortical rust on different 2-needle pines, mainly on *Pinus nigra*, *P. sylvestris*, and *P. halepensis.*

Melampsora pinitorqua Rostr., a heteroecious fungus causing curving and dying back of new pine shoots, mostly on *Pinus nigra* and *P. sylvestris.* Alternate hosts are *Populus alba* and *P. tremula.*

Actinothyrium marginatum Sacc., an agent of red spots on pine needles, mostly on *Pinus nigra* and *P. sylvestris.* The infected needles are shed prematurely.

Cenangium abietis (Pers.) Rehm, a cause of dieback of Austrian and Scotch pine twigs under specific climatic conditions.

Phacidium infestans Karst., the snow blight fungus on Scotch pine, causing death of young plants.

Quercus

Microsphaera alphitoides Griff. & Maubl., well-known oak mildew, concurring in dying back of many oaks, and especially of *Quercus pedunculata.*

Polyporus dryophilus Berk., causing white pocket rot of oak heartwood, with a considerable loss in wood volume.

Loranthus europeus Jacq., a mistletoe with yellow fruits, contributing in oak dying back.

Populus

Dothichiza populea Sacc. & Br., a widespread parasitic fungus, causing necrosis of bark tissue of many poplars, and generally high mortality of 2- to 3-year plants.

Venturia populina (Vuill.) Fabr., producing withering of succulent shoots of black poplars in nurseries and plantations.

Venturia tremulae Aderh., producing similar effects as the preceding organism on *Populus tremula*, *P. alba*, and varieties of the latter species.

Melampsora allii-populina Kleb., a widespread heteroecious fungus, causing rust and premature defoliation of poplar leaves, mainly on species, cultivars, and clones belonging to the section Aigeiros.

"*Brown spot disease*" is to be added although its true agent still remains unknown. The phenomenon is particularly pronounced in euramerican cultivars, but occurs in native poplars also. It spreads continually.

Other work I did was to register those pathogens dangerous to the other native and foreign tree species grown in the south. The recent trend toward the establishment of test plantations of introduced fast-growing species such as pine, oak, larch, and Douglas-fir varieties in a number of southern countries will soon provide better information on the susceptibility of these species to native diseases. Observations until now have been made only in the few existing plantations and sometimes only on the rare individual. The identity of native pathogens found on foreign trees will be included in the completed list of pathogens of southern and eastern Europe.

Czechoslovakia

AUG. KALANDRA

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Only a few different introduced tree species are used in Czechoslovak forestry. Some of the larger forest stands in Bohemia are composed mainly of the introduced *Pinus strobus* which reaches mean or older age. *Larix leptolepis* is grown more frequently but not on a large scale. Introduced spruces are not grown on a large scale either. Douglas-fir has been and still is in high favor. In more recent years *Abies grandis* has been tried. Among the introduced broad-leaved trees, *Quercus rubra* and *Fraxinus americana* are represented here and there. Various North American, European, and Asian species or hybrids of poplar have also been introduced.

Diseases known from other countries are important factors in some regions of Czechoslovakia. *Cronartium ribicola* Dietr. is damaging to *Pinus strobus* here and there in forests, parks, and where used as an ornamental. White pine was formerly favored in the Elbe region of Bohemia because of its resistance to *Lophodermium pinastri*

(Schrad.) Chev. It later became apparent that this species was susceptible to a variety of other diseases, among them the above-mentioned *C. ribicola*, *Armillaria mellea* (Wahl.), and *Leptostroma strobicolla* Hil. Injurious animals increased the damage. For these reasons cultivation of white pine is now limited.

It is worthy of mention that *Cronartium ribicola* occurs individually even on the native *Pinus cembra* in the High Tatra Mountains. Jack pine, *Pinus banksiana*, was cultivated toward the end of the 19th and the beginning of the 20th century because of its resistance to pine needle cast. Low growth rate of this species at older ages caused a return to the use of Scotch pine, *Pinus sylvestris*, despite its susceptibility to needle cast.

European larch, *Larix decidua*, suffers severely from *Dasyscypha willkommii* (Hart.) Rehm. and more resistant races are being sought. The good qualities of the Sudeten and Tatra larch may be stressed rather than trying to use the very susceptible European larch. It is also planned to take advantage of the resistance of *Larix leptolepis* in hybridization work. Larch in forest nurseries is attacked by the needle cast caused by *Meria laricis* Vuill. Experiments should be carried out to test the resistance of various provenances.

Douglas-fir is an especially important forest tree species. *Rhabdocline pseudotsugae* Sydow appeared in the CSSR for the first time in 1938 and is spreading in the existant forest stands of this valuable tree species. Successive attacks on Douglas-fir over a period of several years leads to defoliation and, here and there, to the death of trees. The attacks are quite irregular. This disease has not reduced the popularity of Douglas-fir even though *Rhabdocline* has spread over large areas in various regions of the Czechoslovak Socialist Republic.

Adelopus gaumanni Rohde has not yet been recorded in the CSSR, though it is common in neighboring states (German Democratic Republic, etc.). But Douglas-fir is attacked by the foreign aphid *Gilletteella cooleyi* Gill. In addition, Douglas-fir suffers like other tree species from attacks by the honey fungus, *Armillaria mellea*.

Pinus austriaca is planted artificially on lime and extreme sites. It is more resistant to *Lophodermium pinastri* but more susceptible to *Cenangium abietis* (Pers.) Duby, which has spread in our country notably since 1959. It is also more susceptible to *Naemacyclus niveus* Pers.

Broadleaved tree species are represented notably by *Quercus rubra*. No important diseases are recorded.

Foresters are worried by diseases of poplars grown in pure stands after World War II, and mainly from the year 1955. Our native poplar, *Populus nigra* f. *typica* Schneider, suffers heavily from *Chondroplea populea*, brown spot disease, and other diseases. Imported poplars are not immune, but here and there *Populus monilifera* and Euroamerican poplars suffer less than the native ones.

Hybrids used in forestry and outside the forest, such as *Populus robusta*, *P. regenerata*, and *P. berolinensis*, suffer by brown spotting and *Chondroplea populea*.

Pseudomonas rimaefaciens and *Nectria* do not occur as frequently as in western Europe and other parts of central Europe. *Venturia populina* (Vuill.) Fabr. attacked the poplar, *Populus berolinensis*, in one locality. It is not seriously harmful in other places.

Investigations of recent years show damage to Italian poplar I–214 and other susceptible poplars by late frosts. *Populus simoni* suffers in the CSSR from brown spotting disease and *Dothichiza populea* (*Chondroplea populea* (Jacc.) Kleb.)

Septoria musiva Peck has not yet been found.

Graphium ulmi Schw. damages the elm in forests, parks, and along streets. Foreign elms have not yet been introduced to our country.

Czechoslovak forest management relies mainly upon native tree species. Douglas-fir is the only introduced tree species of considerable economic importance. Exotic tree species and their diseases are given more attention in gardening and park management.

Great Britain

T. R. Peace

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Pressure of existing commitments and staff changes have prevented any substantial developments in forest pathological work in Britain over the last few years. This has inevitably affected our international activities.

Probably the most significant piece of work, internationally, was the organization, under the auspices of IUFRO of a meeting of persons actively interested in the fungus *Fomes annosus*. This was held in Scotland in May 1960, and involved a number of forest excursions as well as meetings; 23 delegates from 9 countries attended including 5 from North America. A report, containing the papers presented by delegates, an account of the field tours, and a summary of the discussions, has been prepared and is now with Dr. Biraghi, Chairman of Section 24, who is dealing with the question of publication.

A list of fungi attacking the genera *Pinus*, *Populus*, and *Quercus* in Britain has been prepared as part of the general scheme. It is my view that before these lists are finalized more thought must be given to the evaluation of the status of the diseases mentioned. The terms used at present are too vague. In addition, value would be added to the list if degree of susceptibility could be mentioned where sound information is available. For instance, *Melampsora pinitorqua* has been recorded on *Pinus nigra calabrica* in Britain, but this record rests on one or two occurrences with negligible damage. Only on *Pinus sylvestris* has it so far proved damaging.

A good deal of the work currently in progress has a definite international interest. Tests of large numbers of poplar clones for their resistance to bacterial canker and the field testing of elms, including some of those produced in Holland, for resistance against *Ceratocystis ulmi* are cases in point. The test area of two-needled pines and aspen poplars to get information on susceptibility to *Melampsora pinitorqua* has yielded some information on American (*Pinus resinosa*, *Populus tremuloides*, *P. grandidentata*) as well as on European species; but it has proved extremely difficult to maintain in an actively infective condition and is being abandoned.

In August and September 1960, J. S. Murray made an extensive tour in Denmark, Germany, and Belgium, visiting Research Stations and inspecting diseases in the forest. During the period in Germany

he received financial help from the West German Bundesministerium, for which we give grateful thanks. This visit resulted in the establishment of many useful contacts, and in valuable exchanges of information.

ACCOMPLISHMENTS: NORTH AMERICA

Canada

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Introduction

The progress made in Canada towards the objectives of the Working Group on International Cooperation in Forest Disease Research, Section 24, the International Union of Forest Research Organizations, is summarized under the following headings: Organization of Forest Pathology in Canada, Forest Disease Surveys, Test Plantations of Exotic Trees, Quarantine Measures, Literature Reviews and Publications of International Interest, and Directory of Forest Pathologists.

The Organization of Forest Pathology in Canada

Research in forest pathology in Canada is undertaken principally by the Forest Entomology and Pathology Branch of the Department of Forestry. Important and significant activities, however, are in progress at other institutions. These agencies are discussed briefly under appropriate headings.

Forest Entomology and Pathology Branch, Canada Department of Forestry.—The Headquarters of the Forest Entomology and Pathology Branch in Ottawa consists of a Director, two Associate Directors, and supporting staff. One Associate Director is responsible for the direction of the national program of research and surveys in forest pathology. Some 60 scientists assisted by approximately 100 laboratory and field technicians are employed at seven regional laboratories located at Victoria, British Columbia; Calgary, Alberta; Saskatoon, Saskatchewan; Maple, Ontario; Quebec City, Quebec; Fredericton, New Brunswick; and Corner Brook, Newfoundland.

Within the Forest Entomology and Pathology Branch close working relationships exist between the pathology and entomology groups, with a high degree of integration and interdependence of personnel, surveys, research investigations, and facilities. In the Forest Insect and Disease Survey program, for example, a number of research officers coordinate insect and disease activities regionally and nationally, and some 75 forest ranger technicians are responsible for field surveys and collections of both diseases and insects. There are joint facilities and personnel for photography, library, biometric guidance, administration, and other services.

A primary function of forest pathology in each laboratory is to conduct a comprehensive program of research and regional surveys of forest diseases with the aim that losses caused by disease may be prevented or reduced to an acceptable level and thereby improve silvicultural and forest management procedures. Close liaison is maintained

with the forest industries, the provincial governments who administrate almost 80 percent of Canadian forests, and other associated agencies. The regional laboratories also contribute technical and advisory services to provincial governments and industries in disease control programs, in the application of pathological procedures to inventory and utilization practices, and in the appraisal of the results of control operations. Instruction in forest pathology is frequently provided to universities and forest ranger schools.

Other Federal Government Departments.—In the Department of Agriculture, the mycology unit of the Plant Research Institute conducts taxonomic studies of fungi and provides identification services. The work of Dr. M. K. Nobles with wood-destroying fungi is of particular interest to forest pathologists. This institute is also responsible for frost resistance studies relating to trees.

In the Forest Products Research Branch, Department of Forestry, research is undertaken at both the Ottawa and Vancouver laboratories on stain and decays of wood products in storage and service.

Provincial Governments.—The provincial forest services provide experimental areas, building sites, and transportation services; their personnel cooperate in forest insect and disease surveys and frequently participate in research projects with the Department of Forestry and other agencies. Forest pathologists will be interested to learn that many provincial governments now obtain information on decays as a regular part of forest inventories, with the Forest Entomology and Pathology Branch staff contributing guidance in field and laboratory techniques.

Universities.—An important contribution of the universities to forest pathology in Canada is the training and development of research personnel. In addition, in forest disease research, Dr. J. E. Bier and his students at the University of B.C. are engaged in studies of bark moisture in relation to the development of canker diseases caused by native, facultative parasites (*1*). The morphology of dwarfmistletoes is being examined by Dr. J. Kuijt, University of B.C. At the University of Toronto, E. Jorgensen is investigating diseases of forest plantations, particularly root rot caused by *Fomes annosus*. At Queen's University, Dr. H. M. Good is concerned with decay organisms of hardwoods and Dr. G. Krotkov is collaborating with Dr. V. Slankis of the Laboratory of Forest Pathology, Department of Forestry, Maple, Ontario, on studies of mycorrhizae.

Research problems of particular interest to the Forest Entomology and Pathology Branch, but requiring facilities not available at regional laboratories, are occasionally conducted by university staff members under Department of Forestry extramural research financial grants. Funds for forest disease research at universities may also be provided by agencies such as the National Research Council, Ontario Research Foundation, and the forest industries.

International Considerations.—Forest pathologists in Canada maintain close contact with their colleagues throughout the world through correspondence and through participation in F.A.O. working groups, various international committees and societies, attendance at world scientific congresses, and by exchange visits with scientists in other countries. Through the Colombo Plan, scientists of member countries are invited by the Canadian Government to engage in study tours of research programs and facilities. At the present time we

are pleased to have Dr. B. K. Bakshi of India making a detailed study tour of forest disease research establishments throughout Canada.

Consultations between Canadian and American pathologists is frequent and three international conferences held annually in the eastern, central, and western regions of Canada and the United States provide excellent opportunities for informal discussions of problems. At the first meeting of the newly formed North American Forestry Commission scheduled for Mexico City, July 24 to July 29, 1961, forest diseases constitute an important part of the agenda of business. This new commission will provide increased opportunities for the mutual exchange of information between pathologists in Canada, the United States, and Mexico and should strengthen the contacts of scientists in North America with those of other countries.

Forest Disease Surveys

The Forest Entomology and Pathology Branch maintains a permanent national organization for continuing surveys of forest diseases and insects. The primary objectives of the forest disease survey are to detect, identify, record, and interpret the significance of tree diseases in Canada.

This survey establishment includes some 75 forest ranger technicians located in the various regions of the country whose duties are to collect and appraise the damage caused by forest diseases and insects. Forest personnel of provincial governments, industries, and other agencies are active cooperators in this program. These activities are coordinated and directed at regional and national levels by research officers. The published results of 10 years (1951–60) work have verified the usefulness of this methodical appraisal of diseases. The results have proved valuable in developing control procedures, in preventing the establishment and spread of new diseases, and in providing a foundation for the planning and development of the disease research program.

Typical of the regional disease survey contributions included in the Annual Report of the Forest Insect and Disease Survey for 1960 (*3*) is the report from British Columbia (*6*). A total of 2,049 disease collections were made from more than 40 tree species. Under "Important Diseases," the following are discussed: weather injury; foliage diseases caused by *Hypoderma deformans*, *H. laricis*, and *Rhabdocline pseudotsugae*; two new diseases introduced from Europe, pine twist rust (*Melampsora pinitorqua*) and pear-juniper rust (*Gymnosporangium fuscum*); willow blight; dying of weeping willow; *Melampsora* rust of Douglas-fir and poplar; diseases of exotic trees; and disease conditions in forest nurseries. Under the title of "Other Noteworthy Diseases," 20 additional diseases are tabulated according to host, organism, locality, and remarks. It is evident that the results obtained from the survey are of interest to pathologists in other countries because many of the pathogens reported have worldwide distribution.

During recent years detailed surveys of native and introduced diseases have included the Dutch elm disease, white pine blister rust, root rots caused by *Fomes annosus* and *Armillaria mellea*, willow blight, the beech-bark disease, various decay fungi, needle cast and rust fungi, poplar canker fungi, dwarfmistletoes and their parasites, and many others.

Special surveys and appraisal studies of diseases of Douglas-fir plantations have been underway for the past few years (*4*). In

Alberta, ground and aerial surveys of dwarfmistletoes on lodgepole pine were initiated in 1960 to improve the methods of survey and assessment of damage.

Compilations of the pathogens of *Pinus*, *Quercus*, and *Populus* have been completed. Six regional reports distributed earlier this year summarize the findings for the extensive regions of Canada covered by each presentation. These data will be collated and revised in one publication. In addition, a comprehensive report on the diseases of *Pinus* with detailed annotations on each pathogen is in an advanced stage of preparation. This will be the first in a planned series of detailed annotated compilations of the diseases of native and exotic trees and should prove of interest to workers in other countries. A series of descriptive publications covering the diseases of major tree species is envisaged and the first of these dealing with lodgepole pine is in preparation.

Test Plantations of Exotic Trees

Numerous species of exotic hardwoods and conifers have been established in plantations throughout Canada during recent years by industries and by provincial and federal governments. Unfortunately, no central agency has the responsibility to collate and ensure the most effective development of these plantings. Also, these plantations have not been grown specifically to determine susceptibility to native diseases. As a result, many plantations may not have been located in the environment most favorable for tree growth and resistance to native diseases. Nevertheless, many of these plantations are yielding data on diseases that are recorded in the Annual Report of the Forest Insect and Disease Survey (*3*).

In British Columbia a central registry for all introduced trees and test plantations in the Province has been set up by the Forest Entomology and Pathology Laboratory, Victoria, and by the B.C. Forest Service (*7*). This development recognizes the dangers involved in the introduction of exotic trees and the objectives are to register all plantations, appraise disease conditions periodically, assess species suitability, and to prevent the introduction of damaging foreign diseases. The exotic plantations under observation now total 168 and are generally of recent origin. The 1960 Survey report (*3*) provides a progress statement on the condition of these plantations. New host-fungus records are tabulated and unfavorable site conditions are suggested to have precipitated a 75-percent incidence of basal canker by *Armillaria mellea* in a plantation of "Robusta" and "Grandis" poplar hybrids (*6*).

In British Columbia, Ontario, and Quebec various studies on the biology and host-parasite relationships of pathogens of hybrid poplars (particularly in the section Leuce) include fungous species in the genera *Cytospora*, *Didymosphaeria*, *Dothichiza*, *Gloeosporium*, *Melampsora*, *Pollaccia*, *Septoria*, *Hypoxylon*, and others. In Ontario and Quebec, special surveys are being conducted in test plantations particularly of hybrid poplars established by provincial governments and industries, and reliable inoculation methods are under consideration in the assessment of host susceptibility. Standardization of inoculation procedures and host material is needed so that results will be meaningful to all countries. In Ontario and British Columbia resistance testing of white pines to white pine blister rust has been

underway for several years and in Quebec tests of resistance of native and European elm hybrids to the Dutch elm disease have been in progress since 1956.

Quarantine Measures

The Destructive Insect and Pest Act (*2*) guards against the introduction and distribution of destructive forest diseases and insects. This Act is administered by the Plant Protection Division of the Department of Agriculture which is empowered to quarantine any foreign disease that has not become widely distributed. Where a disease is well established, the movement of host material may be restricted to prevent the spread of the disease to other parts of the country. Dutch elm disease is an example of a disease that has become permanently established. The importation of all plants and products of *Ulmus* and *Zelkova* is prohibited unless the material has been kiln-dried under specified conditions. To prevent the spread of the disease within the country, the shipment of elm plants or logs from the eastern provinces of Ontario, Quebec, and New Brunswick to other provinces is not permitted.

These regulations are under constant revision according to circumstances. At present, for example, negotiations are under way to include specific clauses in the Act to prevent the introduction and spread of certain diseases such as weeping canker (bacterial canker) of poplar caused by *Pseudomonas syringae*. Suggestions for the improvement of quarantine legislation and procedures are being submitted in the general discussions in Session IV of this symposium.

Literature Reviews and Publications of International Interest

Literature reviews of forest diseases are generally included as part of publications reporting specific research. In addition, some recent reviews include dwarfmistletoes (*5*), the diseases of lodgepole pine (*10*), the diseases of red and white pine (*11*), and general reviews (*8*, *9*). A list of French and English names of tree diseases in Canada is in press. The annotated list of diseases of *Pinus* now in preparation will include the pertinent literature on this subject. Further reviews of forest diseases are planned.

A number of Canadian reports and publications are of interest to pathologists in other countries. The Annual Report of the Forest Insect and Disease Survey has already been mentioned. The Bi-Monthly Progress Report of the Forest Entomology and Pathology Branch is another publication that provides progress statements on research underway at regional laboratories throughout Canada and includes a current list of scientific publications. An annual list of publications of the Forest Entomology and Pathology Branch on forest diseases is also issued and copies of the listed papers, many of which are of international interest, are available upon request, usually without charge.

Directory of Forest Pathologists

A report has been prepared for distribution at this symposium listing the names and research interests of forest pathologists of the Forest Entomology and Pathology Branch, Department of Forestry. In addition to this list of Department of Forestry pathologists, other scientists working in the field of forest pathology in Canada are re-

corded in a separate tabulation. Forest pathologists throughout the world are encouraged to correspond with these scientists.

Summary

This presentation reports on Canada's participation in the Working Group on International Cooperation in Forest Disease Research, as follows:

1. Results from the Forest Disease Survey, which are of international interest, are published each year. Six regional contributions on the diseases of *Pinus*, *Populus*, and *Quercus* have been completed and distributed. A detailed annotated list of diseases of *Pinus* is in advanced preparation and similar presentations are to be compiled for the other major tree species. A series describing the diseases of each important timber tree is also in view. Special surveys are being undertaken on the diseases of Douglas-fir and on dwarfmistletoes of conifers.

2. Quarantine measures are being reviewed.

3. Test plantations of exotic hardwoods and conifers established throughout Canada by provincial governments and industries are being examined periodically to determine susceptibility to native diseases. Inoculation tests are also being made utilizing some of the most important native pathogens. These tests could be made more meaningful to all countries if inoculation procedures and host material were standardized.

4. Literature reviews are generally included as part of each comprehensive publication. Also, a number of reports have been made summarizing the diseases of individual trees or the types of pathogens; more of these reviews are contemplated. Current publications of international interest are listed in the Annual Report of the Forest Insect and Disease Survey and the Bi-Monthly Progress Report. An annual tabulation of publications by the Forest Entomology and Pathology Branch lists a number of research contributions of interest to pathologists in other countries.

5. A directory of forest pathologists and their research activities has been prepared and distributed to IUFRO member countries.

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United States Department of Agriculture

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The U.S. Department of Agriculture has five Divisions—two in the Forest Service, three in the Agricultural Research Service—responsible for activities of direct interest to our Working Group. It would be helpful to characterize briefly the responsibilities of these Divisions.

In the Forest Service, the Forest Disease Research Division plans and carries out investigations to determine the cause of and to formulate control methods for diseases of forest trees and forest products, and the Forest Pest Control Division administers all Federal forest disease control programs, including detection and appraisal surveys. In the Agricultural Research Service, the Crops Research Division plans and carries out investigations to determine the cause of and to formulate control methods for diseases of shade, ornamental, and orchard trees; the Plant Quarantine Division administers all Federal quarantines regulating the importation of plants and plant materials, including their fungus and insect pests; and the Plant Pest Control Division administers similar quarantines regulating the domestic movement of plants and plant materials. These five Divisions, therefore, are responsible for all Federal research and prevention or control programs for tree diseases in the United States. They are headquartered in Washington, D.C., and cooperate closely in carrying out their work.

Progress toward our Working Group objectives by the U.S. Department of Agriculture may be summarized under a few headings: Research, Publications, Quarantines, Exchange of Tree Seed, and Miscellaneous Activities.

Research

In the Department, and primarily in the Forest Service, there are about 100 scientists engaged in some aspect of tree disease research. All major forest tree diseases in the United States are under study to determine their causes, to clarify the taxonomy of pathogens, to establish their diagnostic characteristics in pure culture, to understand the influence of environmental factors on their incidence, and to develop and improve control methods. Control is sought through direct measures such as sanitation or application of fungicides or other chemicals and through indirect measures such as management practices favorable to the tree and detrimental to the disease or selection and breeding for genetic resistance. We also seek to exclude dangerous foreign forest pathogens, a subject I will discuss further under Quarantines.

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Of immediate interest, we have many studies in progress on the principal diseases of *Pinus*, *Populus*, and *Quercus*, several of which will be considered in detail. Examples are as follows:

Diseases of *Pinus* spp.—
Cronartium ribicola, blister rust of white or 5-needle pines;
Cronartium strobilinum, cone rust of southern hard pines;
Cronartium fusiforme, fusiform rust of southern hard pines;
Cronartium comandrae, Comandra rust of western hard pines;
Peridermium harknessii, gall rust of western hard pines;
Scirrhia acicola, brown-spot needle blight of longleaf pine;
Elytroderma deformans, needle blight of ponderosa pine; and
Fomes annosus, root rot of conifers.

Diseases of *Populus* spp.—
Hypoxylon pruinatum, Hypoxylon canker of aspen.

Diseases of *Quercus* spp.—
Ceratocystis fagacearum, oak wilt.

In addition, we are investigating the heart rots of all species to determine the causal fungi, how they enter living trees, how rapidly they decay wood, and the relation of rot volume to tree age, vigor, and site conditions. For all western conifers we are studying the factors that regulate infection and intensification of the dwarfmistletoes, *Arceuthobium* spp., which are a major cause of loss in forest productivity. For many diseases, particularly the *Cronartium* rusts and the dwarfmistletoes, we are strengthening our research to develop controls with systemic chemicals.

Publications

Federal research on forest tree diseases in the United States has been carried on continuously since 1899. By the end of 1960 about 2,800 articles had been published on the cause and control of diseases of forest trees and forest products. These were published as government bulletins, in scientific journals, and in a wide variety of technical, trade, and popular publications. Titles through 1953 are listed in USDA Miscellaneous Publication No. 725, "Bibliography of Forest Disease Research in the Department of Agriculture." We plan to publish additions to the bibliography at 10-year intervals. In the meantime we issue annual lists of publications such as the one for 1960, available for distribution at this Congress. Instructions on how to request copies of articles are attached. With few exceptions, publications of the U.S. Department of Agriculture are available at no cost.

Of the many recent publications on forest diseases issued by the U.S. Forest Service, three by Dr. Perley Spaulding are of particular significance to this Congress. They are USDA Handbooks Nos. 100, 139, and 197, respectively published in 1956, 1958, and 1961. No. 100 is titled "Diseases of North American Forest Trees Planted Abroad"; No. 139, "Diseases of Foreign Forest Trees Growing in the United States"; and No. 197, "Foreign Diseases of Forest Trees of the World."

In 1955 we started a new series of publications known as Forest Pest Leaflets. To date, 54 leaflets have been issued, of which 26 are on diseases and 28 are on insects. These leaflets summarize for popular

use available information on the cause, hosts, symptoms, distribution, and control of the subject disease or insect.

Quarantines

In 1912 the Federal Plant Quarantine Act was enacted in the United States to prevent the importation of plant pests. Under this Act there are prohibitions and limitations regulating the importation of woody plants—seed, seedlings, cuttings, or any plant part—that may result in the entry into the United States of injurious plant diseases and insects. The importation of 53 woody genera, including 22 genera of forest trees, is prohibited except as seed or for experimental use under rigid restrictions. No plant pest—"insects, nematodes, bacteria, fungi, other parasitic plants or reproductive parts thereof, viruses, or any similar organisms or infectious substances which can cause disease or damage to plants or plant products"—may be imported from abroad without authorization under a general or specific permit issued by the Department of Agriculture. Under the provisions of this Act, our inspectors, in 1960, intercepted and destroyed about 31,000 potentially destructive plant pests, one every 17 minutes. I have no figures on how many of these interceptions were of potential forest pests.

In 1960 the U.S. Forest Service formally approved two policies of particular interest to this Congress—one with reference to importation and the other to exportation of woody plants or parts thereof. We will import forest trees or other plants into the United States by seed only, subject to postentry fumigation, except in those cases where the plant cannot be reproduced adequately or exactly by seed, as in clonal lines. In such cases, quarantine regulations will be rigidly observed. We will export treated seed only as a means of introducing U.S. trees or plants into foreign countries, except in those cases where the plant cannot be reproduced adequately from seed. In such cases, we will inform the importer of the hazards involved and recommend adequate precautions, such as inspection, fumigation, and isolation.

Exchange of Tree Seed

The Forest Service cooperates with foreign scientific institutions, universities, and public agencies in the collection and exchange of small quantities of forest tree seeds and other forest plant material for research purposes. In 1960, for example, we received 116 requests from 39 foreign countries for 603 collections of U.S. forest tree seed. We have filled 337 of these requests and will handle most of the remaining ones in the near future. Also in 1960 we had 19 requests from within the U.S. for 226 collections of foreign forest tree seed, of which 135 have been filled. Involved in these exchanges were seed from one to several species of important forest trees in 24 genera. If this rate of exchange of tree seed continues, or increases as it has in the past few years, and the introduced species are established in plantations, it should provide excellent opportunities to determine the susceptibility of many exotic trees to the diseases of other continents.

In making these exchanges we adhere to the policies on importation and exportation of tree seed as set forth in the preceding section.

Miscellaneous Activities

Special surveys and studies are in progress to determine the relative susceptibility of native and exotic forest tree species to diseases in the U.S. Among such diseases are white pine blister rust (*Cronartium rib-*

icola), fusiform rust (*C. fusiforme*), brown-spot needle blight (*Scirrhia acicola*), annosus root rot (*Fomes annosus*), Hypoxylon canker (*Hypoxylon pruinatum*), oak wilt (*Ceratocystis fagacearum*), and dwarfmistletoes (*Arceuthobium* spp.).

Selection and/or breeding to improve genetic resistance to specific forest tree diseases is underway in the following genera:

Castanea for resistance to *Endothia parasitica;*
Populus for resistance to *Septoria musiva;*
Ulmus for resistance to *Ceratocystis ulmi* and *Morsus ulmi;*
Juniperus for resistance to *Exosporium glomerulosum* and *Phomopsis juniperovora;*
Pinus for resistance to *Arceuthobium* spp., *Cronartium ribicola*, *C. fusiforme*, and *Phytophthora cinnamomi;* and
Pseudotsuga for resistance to *Rhabdocline pseudotsugae.*

In addition, for all forest tree diseases our pathologists have been directed to note any instances of apparent resistance and to take action as needed to insure that the germ plasm is not lost.

A recent development in the United States will stimulate increased international cooperation in forest disease research. The United States sells surplus agricultural commodities—wheat, cotton, and tobacco, for example—to many countries. Payment for these commodities is accepted in the currency of the recipient country and has resulted in the accumulation of considerable U.S. credit balances in some countries. In 1958 we were given the authority by Congress to use a portion of these funds for agricultural and forestry research of benefit to the United States. Since that time we have negotiated 234 foreign research projects, including 43 in forestry distributed as follows: Europe 27, Asia 15, and South America 1. Five of the forestry projects are in forest disease research. Two are on the role of mycorrhizae in tree nutrition, growth, and disease susceptibility (in Finland and Poland); one is a survey of the diseases of native and exotic conifers (in Spain); another is on the antibiotic relationship of saprophytic soil fungi to forest tree root pathogens (in Poland); and the remaining one is a study of the susceptibility of North American forest tree species to insects and diseases (also in Poland). We are currently negotiating similar susceptibility studies in Columbia and Uruguay, S.A. These research projects will contribute directly to our Working Group objectives.

In conclusion, I would like to say that we in the U.S. Department of Agriculture will take advantage of every opportunity to carry out research at home and abroad that will provide a better understanding of the nature and cause of destructive forest diseases and will lead to improved methods of reducing disease losses.

United States

A. J. Riker

Professor of Plant Pathology and Forestry, University of Wisconsin, Madison, Wisconsin, U.S.A.

Studies are under way at Wisconsin on some potentially dangerous diseases.

Oak Wilt

If oak wilt should suddenly appear in other continents, some of the following control measures might be worth considering. When the infected tree and all other trees within 10 to 15 meters are killed with sodium arsenite, local spread of the disease is stopped. Sodium arsenite must be used with suitable precautions so that animals are not poisoned. This chemical has been effective, but many others have been inadequate or worthless. Where desirable, the local spread may be prevented by cutting the roots between infected and healthy trees or by placing a chemical barrier between healthy and diseased trees with methyl bromide or Vapam injected in the soil. A girdle through at least the last three annual rings may hasten the desiccation of the tree and reduce fungus mat formation.

Poplar Cankers

Natural and artificial poplar hybrids are under test for resistance to various diseases. Cankers caused by *Hypoxylon*, *Dothichiza*, *Septoria*, and *Fusarium* are among the more important. In addition, attention is being given to such problems as wet wood, spring defoliation, rust, and dieback.

Some of these fungi are well known in Europe and elsewhere. However, we do not yet know whether they are the same races as those which occur in North America.

White Pine Blister Rust

In addition to the conventional control of white pine blister rust by the eradication of *Ribes* bushes, research is underway to develop resistant white pines. First generation progenies from controlled crosses with six resistant selections contain a considerable percentage of resistant seedlings.

The incidence of white pine blister rust infection in Wisconsin is associated with the microclimate. Silvicultural aids to control can be made on the basis of forest stand microclimates. Descriptions of local climatic situations have been worked out that serve as guides for removal of *Ribes* bushes only when necessary. They serve also for selecting sites for planting white pine. In some places the danger from the blister rust is great; in others the danger is too small to consider. Such areas may be quite close together. Scions and seed from elite white pines have been sent to a number of places abroad.

Dutch Elm Disease

Research is underway to reduce the damage from the Dutch elm disease through genetic resistance and with systemic chemicals both against the fungus and against the insect vectors.

Seed Collections

At the request of Dr. Takahashi, seeds from various Wisconsin species have been collected and forwarded to Japan.

The research reported has been done in cooperation with the Wisconsin Conservation Department, the U.S. Forest Service, the Nekoosa Edwards Paper Company, and others.

Summary of Conferences Abroad

In 1959–60 Professor A. J. Riker consulted with authorities in North America, Europe, Southeast Asia, and the South Pacific on what should be done about internationally dangerous tree diseases. He went on a Haight Travelling Fellowship from the University of Wisconsin. His study was a part of the work of Section 24 of the International Union of Forest Research Organizations. A summary of the results of his discussions provides the basis for the fourth and last session of our Working Group.

THE THREAT OF INTRODUCED DISEASES

T. R. Peace

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Relatively few fungi, bacteria, and viruses attacking forest trees have a worldwide distribution, or even cover all the areas where availability of host and suitability of climate would permit their development. Many instances are already known, however, where pathogens have spread from one continent to another, and there are alarming possibilities of further spread. This paper attempts to discuss the risk, and how its immediate and long-term effects can be lessened. Discussion is limited to diseases caused by fungi, bacteria, and viruses, but similar principles apply to insect pests.

On land masses where the host tree or trees have a continuous distribution, spread of diseases by natural means is almost inevitable and extremely hard to stop. But where there are breaks in host distribution, for instance a mountain range or a desert, and particularly where there is a sea barrier, natural dissemination becomes much less important or even nonexistent, and man is the chief agent of spread (Orton and Beattie 1923). Thus intercontinental transmission of disease, except between Europe and Asia, is almost entirely due to man's agency.

This paper is divided into two main sections. In the first and smaller, the risk is considered briefly. In the second, various actions, which have already been taken or can be taken in the future, are considered. The second part may perhaps be regarded as outside the proper purpose of the paper I was asked to submit. If so, I must apologize to Professor Boyce on whose ground I will have trespassed. Nevertheless it may be valuable if we have these actions in mind when we are considering individual diseases. In any case, this is a matter in which I have some experience and rather definite views. This leads me to welcome an opportunity of putting them before the Group.

The Problem

A study of the present known distribution—both in the positive sense of areas known to be infected and in the negative one of areas known to be uninfected—makes it clear that many pathogens already known to be dangerous are not yet generally present throughout their host's geographical range. In particular, they may not yet have penetrated all the areas where the host species has been planted as an exotic. Bacterial canker of poplar, whether it is caused by *Pseudomonas syringae* or *Aplanobacterium populi*, is still apparently confined to Europe; *Phloem necrosis*, a virus disease of elm, to North America; the fungus *Monochaetia unicornis*, causing canker on cypresses, to Africa and New Zealand; and *Ceratocystis fagacearum*, the cause of oak wilt, to North America. The compilation of any list of dangerous

pathogens of incomplete distribution indicates clearly that the incompleteness rests partly on lack of knowledge.

A recent unpublished list of forest diseases considered potentially dangerous in Britain was based on reasonably certain information that the diseases listed were not present in this country, but that they were present in other areas. Most records were from North America and Europe, where forest pathological investigations have been reasonably thorough. The tabulation below, based on this list, shows the number of diseases, absent from Britain but considered potentially dangerous to British forest trees, recorded from the different continents:

Country	*Number*
North America only	40
Europe only	8
Asia only	3
North America and Europe	5
North America and Asia	1
North America, Asia, and Europe	2
North and South America	2
North and South America and Europe	1
North and South America, Europe, and Asia	1
Europe and Asia	1
Africa and New Zealand	1

The preponderance of North American entries in this list is partly due to the importance of North American conifers in British forestry. The relatively small number of dangerous European diseases, not present in Britain, is partly the result of its proximity to the rest of Europe, so that many diseases have already achieved a common distribution. The low numbers for other continents are mainly due to the relatively small importance of their trees in Britain and to the fact that in some cases large areas lie in entirely different climatic zones from Britain. Nevertheless, it is almost certain that the low figures for Asia, where nearly all the dangerous diseases listed are Japanese records, and for other continents poorly represented in the list, are also partly due to lack of knowledge of the pathogens occurring there.

While any list of known dangerous pathogens with incomplete continental distributions is certainly impressive, particularly in the number of species involved, it is certain that more diseases remain to be discovered when pathologically unexplored regions, such as China or most of the temperate area of South America, are properly investigated.

Thus, we are faced with a large number of diseases known to be damaging in the countries where they are present and therefore presumed to be damaging in other countries to which they might spread. There is the strong possibility that other diseases, not serious in their present range, might prove dangerous if transported to other areas, either more favorable to their activities or less favorable to their host tree. Finally, there are almost certainly parasites, as yet unrecorded, that will attract attention only when the countries where they now exist are pathologically explored, or when they reach other countries. Thus, we must consider what immediate steps can be taken to prevent or slowup disease spread from place to place, and what

long-term work can be undertaken to mitigate the effects of such diseases should they eventually reach other countries or other continents.

Partial Solutions

Phytosanitary Action

Phytosanitary action can be taken in a number of ways. General reviews of these have been made by Güssow (1936), McCubbin (1946, 1954), and Soraci (1957). The means used fall roughly into three categories: (a) measures taken in the exporting country, (b) measures taken in the importing country, and (c) embargoes, whereby carriage of specified plants or parts of plants from one country to another is completely forbidden.

As far as trees are concerned, there are three main types of material on which fungi, bacteria, and viruses are likely to be transported, namely seed, plants, and timber. Seeds present the smallest risk. Although a good case can be made for the routine treatment of many agricultural seeds, there is so little evidence of disease transmission on tree seeds that no general action can be justified. Only in the case of chestnut blight, *Endothia parasitica*, on *Castanea* seed is there a strong case for seed disinfection.

Timber is much more dangerous, especially when it is unbarked. *Ceratocystis ulmi*, the cause of elm disease, was taken from Europe to North America on unbarked timber, and *Endothia parasitica* may have reached Europe from North America in the same way. There has been a very general tendency, except in the case of fruit and vegetables, to neglect the risk involved in the movement of final products, partly no doubt because restrictions on them would almost invariably involve serious restrictions on trade. Yet transmission of some pathogens, such as *Endothia parasitica* on *Castanea* and on *Quercus*, or *Hypoxylon pruinatum* on poplar, is much more probable on timber than on small plants. Morgan and Byrne (1957) have recently stressed the dangers inherent in the uncontrolled movements of timber from country to country or from continent to continent.

There are of course several well-known examples of the transmission of tree diseases on young plants. The most notable is the importation on nursery plants of *Cronartium ribicola*, the cause of white pine blister rust, into North America from Europe. Most of the quarantine regulations designed to prevent the entry of tree diseases have referred to living plants, rather than to seed or timber.

Although embargoes obviously give the best chances of success, there is rather general objection to their use. Where trade exists they are bound to be restrictive, and there is always the possibility that they may be applied for economic rather than phytosanitary purposes. A complete embargo on seed would obviously impose too severe a restriction on any country which was largely dependent on exotic trees. A complete embargo on young trees and parts of trees would stop the international distribution of trees such as poplars, which are normally raised as vegetatively propagated clones and of grafting material of selected trees for breeding work. A complete embargo on timber would only be possible for a country that possessed a sufficiency of all kinds of timber for its own use. Conditions vary in different countries and, therefore, so does the extent to which they

are able to apply embargoes, undeniably the most effective phytosanitary measures. In practice, embargoes can hardly be made complete. Limited importation by licenses subject to specified precautions must be granted for scientific purposes. In any case spores may enter on aircraft, and passengers may carry diseased material knowingly or unknowingly in their baggage (Sherman 1957).

It is generally admitted that inspection of imported material on arrival is a very unreliable way of preventing the entry of diseases (Gravatt and Parker 1949; Gram 1955). In the case of large consignments only a sample can be inspected and, unless the incidence of disease is very high, it may well go undetected. In some cases the disease may be in a stage which is not detectable even by careful visual inspection. For instance, *Chrysomyxa rhododendri* was imported into the United States as mycelium in azalea leaves. By the time fructifications appeared and the disease became detectable, the plants had been widely distributed (Gould, Eglitis, and Doughty 1955). In fact, inspection on entry, except for the occasional detection of very badly diseased consignments or of material subject to embargo, is valuable mainly in encouraging better phytosanitary practices in the exporting country.

There is obviously a better chance of achieving satisfactory inspection in the country of origin. If plant inspection is started in the nursery, it can be extended over a period of time and carried out with a knowledge of some of the possibilities of infection to which the crop is subject. Even then it is impossible to state honestly that the plant material is entirely free of all disease. There is no doubt, however, that efficient preexport inspection, especially in the nursery, can greatly lessen the risk so that the danger of importing from any particular country depends not only on the pathogens present there, but on the efficiency of its phytopathological inspection service. Every country is therefore in a position to lessen the chances of disease transmission by improvements in the scope and skill of its inspection service and by the encouragement of phytosanitary practices among its exporting nurserymen.

The principles underlying plant quarantine regulations have been discussed by Moore (1952, 1955) and by Soraci (1957). There is a strong tendency to base regulations on our present knowledge of specific diseases, in particular on their potential danger and on their distribution, and to condemn widespread embargoes on the ground that they are based on unreasonable fears of the unknown. However, we must remember our almost complete lack of knowledge of potential tree pathogens in some of the phytopathologically less advanced countries. It is also necessary to take into account the obvious possibility that a known pathogen may behave quite differently when moved to a new environment. The behavior of *Phaeocryptopus gäumannii* on *Pseudotsuga* and *Keithia thujina* on *Thuja*, when moved from North America to Europe, are frightening examples; however, the increased importance of *Keithia* is due mainly to its severe attacks on nursery plants of *Thuja* in Europe, as compared with its behavior on natural regeneration in North America.

Even when a fungus is already widespread, there is a danger that it may have regional strains of differing virulence, the transfer of which could prove damaging. This may well be the case with *Lophodermium pinastri*, the needlecast of pines. Such considerations may support the

application of embargoes where they can be imposed without damage to the forest economy. In the author's opinion, the embargo should always be considered when phytosanitary measures are being devised, and only dismissed on very strong reasons against its use.

It is outside the scope of this paper to discuss all the quarantine measures which have been imposed by various countries against tree diseases. They have been fully summarized in an F.A.O. publication by Ling (1952 and after). The United States regulations, with a very long list of tree genera and species, and with some restrictions applying only to specified regions, represent a painstaking effort to relate quarantine measures to the existence and distribution of known diseases. The British regulations, as far as conifers are concerned, provide an example of the widespread embargo designed to protect a group of genera from all pathogens known or unknown from all regions.

In any case, of course, phytosanitary measures can only postpone the more or less inevitable spread of all diseases to all areas where there are susceptible crops for them to attack. Nevertheless such a postponement may have enormous value in giving time for resistant strains to be discovered or developed, and for control measures to be elaborated.

The remaining sections of this paper indicate what can be done during this "period of grace" to ensure that we are equipped to meet the forest pathological problems of the future when transport will be so rapid and so cheap that restrictions on movements of goods will be completely out of place.

Exploratory Action

When we consider tropical forests, it is readily apparent that the area which is relatively unexplored from the disease point of view is very large. For many extensive regions incomplete lists of fungi do exist; but rarely, except in the more highly developed countries, have the listed fungi been evaluated phytopathologically. Thus, we have little idea how much damage they are doing in their native country and no information at all on which to base estimates of their behavior if they were moved to other countries or continents. Obviously, therefore, there is a great need for what might be called "phytopathological exploration," not only in many tropical forests, but also in quite large areas in the temperate regions. It is clearly important that such work should be done thoroughly and critically. First, an important disease may exist outside the forest proper on scattered trees or in scrub areas, so that surveys cannot be limited to areas of utilizable forest. Second, diseases may be of slight importance in their country of origin because the native host trees are either resistant, as was the case with *Endothia parasitica* on *Castanea* in Asia (Beattie and Diller 1954), or of restricted distribution.

Clearly it is difficult, and in the case of tropical forests impossible, to assess the risk exactly or to consider general precautionary measures until we have a much more widespread knowledge of tree diseases. Unfortunately, while short-term visits such as collecting expeditions may provide valuable information on the occurrence of pathogens, evaluation of these pathogens requires study over a period of time and must therefore await the development of a forest pathological organization in the region concerned.

Research Action

Any advance in our knowledge of tree diseases is naturally of value when we come to consider the possibilities and results of their transmission from one area to another. The testing of the trees of one country or continent for their resistance or susceptibility to the disease of another, whether by chance exposure, deliberate exposure, or inoculation, is particularly valuable. In many countries there is so much information already available, but not necessarily collected, from existing plantations of exotics that it is doubtful if plantings designed merely for disease recording purposes can be justified. These test plantings are really only useful when placed in regions where the pathogen population is unexplored. The possibility of doing this is, of course, usually limited by the lack of pathologists in those regions. It may therefore prove easier to observe the development of diseases in carefully sited plantations, rather than over the unexplored area as a whole.

In addition, forest plantings of exotics are probably lacking in a relatively undeveloped country, so that stands specially formed for pathological study may provide the only chance of getting data on the susceptibility of nonnative trees to the local pathogens. Provided the limitations imposed by siting in relation to virtually unknown sources of disease and by the limited number of plantations are taken into consideration, there is no doubt that such special plantations could yield results of very definite international value.

In pathologically explored regions, plantations designed to test under natural conditions the reactions to specific diseases and therefore deliberately placed in regions of high infectivity would be more valuable, and there is certainly room for their extension. It is also desirable to extend inoculation tests in one country on tree species of importance to another (Gravatt and Parker 1949; Riker 1957).

With any of these methods, care must be used in the interpretation of results. The presence of a disease in a plantation is always more significant than its absence; very good evidence is required before the absence of a disease from a tree can be taken by itself as proof of immunity or resistance. Even when disease is present, care must be exercised, for unequal distribution of sources of infection may cause unequal distribution of damage, which thus may have no relation to the inherent susceptibility and resistance of the trees affected. For this reason, information from a large number of exotic plantations, preferably growing under a wide range of infective and climatic conditions, is likely, if intelligently interpreted, to be of more value than that collected from a limited number of specially designed test plantations.

None of the methods mentioned above will eliminate the effect on diseases of differences in climate and other environmental factors between one country and another. For this reason, the disease reactions of a tree species in one place cannot be taken as a certain indication of its behavior towards the same pathogen elsewhere. They do, however, give the best information which can be procured without actually moving the pathogen, a method which naturally is completely inadmissible.

There is danger even in moving cultures of pathogens from one country to another (Wheeler 1957). Transfer of cultures can be justified only if it serves some useful purpose and provided the cultures

are carefully guarded. The use of an exotic pathogen, or even of an exotic strain of a native pathogen, for inoculation purposes is a pathological crime of grave significance.

There is also room for research on the means of transmission of tree diseases over long distances, though this is of less permanent value than work on disease behavior. Too little is known about the relative importance of different means of transmission. In particular it would be valuable to have more evidence on the importance of seed as a means of transmission.

Evaluatory Action

Any increase of knowledge of the distribution of diseases and of their behavior on different hosts in different climates is of great assistance in evaluating their importance, both present and potential. If more facts were available on the actual and potential economic losses inflicted by diseases transported from one area to another, there would be more reasoned support for expenditure, either on means to prevent their spread or on the mitigation of damage once they had done so. In the past there has been a tendency to behave as if all damaging diseases were of equal importance, at any rate as far as restrictions of spread is concerned. It might be better if our energies were concentrated on the restriction of a limited number of diseases, leaving others to spread more or less unhampered, either because they were easily controlled or avoided, or because there was good evidence that they would not be seriously damaging. However, selective action of this kind would be possible only on the basis of much greater knowledge of disease behavior than we yet possess for most forest pathogens. It can therefore only be practiced when much more work has been done on what might be termed "international disease behavior."

Practical Possibilities

It is apparent from what has been said above that the amount of work required to build up a knowledge of disease behavior, sufficient to form a basis for international action is very great indeed. The limitations on this work are primarily those of staff. At first sight the most important limitation is the absence of forest pathologists in many countries, but the very small number in most other countries, particularly European countries, is equally serious. In many cases, the workers are so few that their energies must inevitably largely be devoted to immediate forest pathological problems. They have little time to spare for investigations which are not of immediate practical value or which are designed with long-term international, rather than immediate national, ends in view. Most governments take the view that forestry, earning as it does low rates of interest, cannot support a high expenditure on research. And thus they expect any substantial increases in research expenditure to be clearly linked with resultant economic advantages. Economic appraisals of disease losses, and in particular of the relation of research costs to the prevention or mitigation of these losses, are thus obviously desirable. Only thus can a reasoned case be stated for increased expenditure on pathological research.

In the meantime, any schemes for international cooperation must either take full account of the limitations which the preoccupation of forest pathologists with their local problems imposes, or else allow

money for the appointment of special staff to carry out the work envisaged. In the field of international cooperation, it is perilously easy to envisage grandiose schemes, the initiation, let alone the operation, of which is far beyond the means available. Of course the fullest possible international use should be made of work already in progress, but even the coordination of this involves considerations of time and money, which cannot be overlooked.

Thus, while few would deny the importance of increasing our knowledge of diseases, of the damage which they are likely to do if they become more widespread, and of the means by which this damage can be avoided, it is vital that the international work involved should be provided with a sound economic background, thus justifying the substantial expenditure involved.

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EXAMPLES OF WIDELY DISTRIBUTED FOREST DISEASES

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In our review of selected dangerous forest diseases in Asia, Europe, and North America, our primary objective is to call attention to a few diseases whose importance in these continents illustrates their potential damaging capacity if they should be introduced to other continents. Most of them are presently restricted to one continent but we have included a few that have already become well-established abroad. In all cases, however, precautions are recommended to prevent or limit further spread to additional continents.

There are many destructive forest diseases already widely distributed. This does not mean that further efforts are unnecessary to restrict the spread of the pathogens that cause them. Many fungi have two or more races, sometimes varying in pathogenicity. For example, *Puccinia graminis* Pers., the cause of stem rust of cereals and grasses, has been differentiated into at least six distinct varieties in North America, varying in spore size and the kinds of plants that they attack. Within these recognizable varieties there are races that may be indistinguishable by morphological characteristics but differing markedly in pathogenicity. There is nothing to preclude the existence or the development of similar races within species of destructive forest fungi. For that reason, measures to prevent further spread of widely distributed forest diseases are important.

The following forest diseases are known to occur on two or more continents. The list is not exhaustive but includes many well-known, widely distributed forest pathogens. The tabulation uses the following abbreviations: Af., Africa; As., Asia; Aus., Australia or New Zealand; E., Europe; NA, North America; SA, South America; W, Worldwide. The pathogens and their distribution are:

Pathogen	*Distribution by Continents*
Agrobacterium tumefaciens (Sm. & Town.) Conn	W
Armillaria mellea (Vahl.) Quel	W
Ceratocystis ulmi (Buism.) Moreau	As., E., NA
Coryneum cardinale Wag	Af., Aus., E., NA
Cronartium ribicola Fisch	As., E., NA
Diplodia pinea (Desm.) Kickx	W
Dothichiza populea Sacc. & Briard	E., NA, SA
Endothia parasitica (Murr.) Ander	As., E., NA
Fomes annosus (Fr.) Cke	As.,E., NA
F. pini (Fr.) Karst	As., E., NA
Fusicladium saliciperdum (All. & Tub.) Tub	E., NA
Lophodermium pinastri (Fr.) Chev	As., Aus., E., NA
Phaeocryptopus gaumanni (Rhode) Petr	E., NA
Physalospora miyabena Fuk	As., E., NA
Phytophthora cinnamomi Rands	As., Aus., NA
Rhabdocline pseudotsugae Syd	E., NA
Valsa Kunzei Fr	As., E., NA
Verticillium albo-atrum Rein. & Bert	Af., Aus., E., NA

SELECTED DANGEROUS FOREST DISEASES IN ASIA, EUROPE, AND NORTH AMERICA

Summary by Host and Continent

The following tabulation summarizes dangerous pathogens by host and distribution. Abbreviations used are: Eur., Europe; N.A., North America; S.A., South America.

Host, family, and genus	*Pathogen*	*Symptoms*	*Continents*
Acanthaceae: *Ruellia*	*Cronartium flaccidum*	Leaf rust	Asia, Eur.
Aceraceae:			
Acer	*Helicobasidium mompa*	Root rot	Asia
	Strumella coryneoidea	Canker	N.A.
Anacardaceae: *Rhus*	*Helicobasidium mompa*	Root rot	Asia
Asclepiadaceae:			
Asclepias	*Cronartium flaccidum*	Leaf rust	Asia, Eur.
Cynanchum	do	do	Do.
Balsaminaceae: *Impatiens.*	do	do	Do.
Betulaceae:			
Ostrya	*Clitocybe tabescens*	Root rot	N.A.
	Strumella coryneoidea	Canker	Do.
Bignoniaceae:			
Catalpa	*Helicobasidium mompa*	Root rot	Asia
Chilopsis	do	do	Do.
Casuarinaceae:			
Casuarina	*Clitocybe tabescens*	do	N.A.
	Trichosporium vesiculosum	Wilt	Asia
Cornaceae:			
Cornus	*Clitocybe tabescens*	Root rot	N.A.
Nyssa	*Strumella coryneoidea*	Canker	Do.
Cupressaceae:			
Callitris	*Clitocybe tabescens*	Root rot	Do.
Chamaecyparis	do	do	Do.
	Helicobasidium mompa	do	Asia
	Phomopsis juniperovora	Needle blight.	N.A.
	Poria weirii	Root rot	Do.
Cupressus	*Clitocybe tabescens*	do	Do.
	Phomopsis juniperovora	Needle blight.	Do.
Juniperus	*Clitocybe tabescens*	Root rot	Do.
	Phomopsis juniperovora	Needle blight.	Do.
	Phymatotrichum omnivorum	Root rot	Do.
Thuja	*Clitocybe tabescens*	do	Do.
	Helicobasidium mompa	do	Asia.
	Keithia thujina	Needle blight.	Eur., N.A.
	Phomopsis juniperovora	do	N.A.
	Phymatotrichum omnivorum	Root rot	Do.
Dipterocarpaceae:			
Shorea	*Fomes caryophylli*	do	Asia.
	Hypoxylon mediterraneum	Canker	Do.
	Polyporus shorea	Heart rot	Do.
Ebenaceae:			
Diospyros	*Cephalosporium diospyri*	Wilt	N.A.
	Phymatotrichum omnivorum	Root rot	Do.

Host, family, and genus	*Pathogen*	*Symptoms*	*Continents*
Eleagnaceae:			
Eleagnus	*Phymatotrichum omnivorum*	Root rot	N.A.
Ericaceae:			
Arctostaphylos	*Chrysomyxa arctostaphyli*	Leaf rust	Do.
Fagaceae:			
Castanea	*Ceratocystis fagacearum*	Wilt	Do.
	Cronartium fusiforme	Leaf rust	Do.
	C. quercuum	do	Do.
	Helicobasidium mompa	Root rot	Asia.
	Pseudomonas castaneae	Blight	Do.
	Strumella coryneoidea	Canker	N.A.
Castanopsis	*Ceratocystis fagacearum*	Wilt	Do.
	Cronartium fusiforme	Leaf rust	Do.
Fagus	*Strumella coryneoidea*	Canker	Do.
Lithocarpus	*Ceratocystis fagacearum*	Wilt	Do.
	Cronartium fusiforme	Leaf rust	Do.
Quercus	*Ceratocystis fagacearum*	Wilt	Do.
	Clitocybe tabescens	Root rot	Do.
	Cronartium fusiforme	Leaf rust	Do.
	C. quercuum	do	Do.
	Helicobasidium mompa	Root rot	Asia.
	Strumella coryneoidea	Canker	N.A.
Gentianaceae:			
Gentiana	*Cronartium flaccidum*	Leaf rust	Asia, Eur.
Ginkoaceae:			
Ginko	*Helicobasidium mompa*	Root rot	Asia.
Hamamelidaceae:			
Liquidambar	*Clitocybe tabescens*	do	N.A.
Hippocastanaceae:			
Aesculus	*Guignardia aesculi*	Leaf blotch	Eur., N.A.
Juglandaceae:			
Carya	*Clitocybe tabescens*	Root rot	N.A.
	Strumella coryneoidea	Canker	Do.
Juglans	*Helicobasidium mompa*	Root rot	Asia
	Phymatotrichum omnivorum	do	N.A.
Platycarya	*Helicobasidium mompa*	do	Asia
Leguminosae:			
Acacia	*Clitocybe tabescens*	do	N.A.
	Fomes badius	Heart rot	Asia
	Glomerella acaciae	Anthracnose	Do.
Albizzia	*Clitocybe tabescens*	Root rot	N.A.
Caragana	*Phymatotrichum omnivorum*	do	Do.
Cercis	*Helicobasidium mompa*	do	Asia
Dalbergia	*Fusarium solani*	Wilt	Do.
Gleditsia	*Chlorogenus robiniae*	Virus	N.A.
	Phymatotrichum omnivorum	Root rot	Do.
Gymnocladus	do	do	Do.
Robinia	*Chlorogenus robiniae*	Virus	Do.
	Helicobasidium mompa	Root rot	Asia
	Phymatotrichum omnivorum	do	N.A.
Liliaceae:			
Allium	*Melampsora allii-populina*	Leaf rust	Eur.
Loascaceae:			
Grammatocarpus	*Cronartium flaccidum*	do	Asia, Eur.
Loasa	do	do	Do.
Magnoliaceae:			
Liriodendron	*Helicobasidium mompa*	Root rot	Asia
Moraceae:			
Broussonetia	do	do	Do.
Ficus	*Clitocybe tabescens*	do	N.A.
	Helicobasidium mompa	do	Asia
Maclura	*Phymatotrichum omnivorum*	do	N.A.
Morus	*Helicobasidium mompa*	do	Asia
	Phymatotrichum omnivorum	do	N.A.
Myrtaceae:			
Eucalyptus	*Clitocybe tabescens*	do	Do.
Oleaceae:			
Fraxinus	*Pseudomonas fraxini*	Canker	Eur.
	Phymatotrichum omnivorum	Root rot	N.A.

Host, family, and genus	*Pathogen*	*Symptoms*	*Continents*
Pinaceae:			
Abies	*Arceuthobium campylopodum.*	Witches'-broom.	N.A.
	Phomopsis pseudotsugae	Canker	Eur.
	Poria weirii	Root rot	N.A., Asia
	Rehmiellopsis balsameae	Needle blight.	N.A.
Cedrus	*Clytocybe tabescens*	Root rot	Do.
	Peridermium cedri	Witches'-broom.	Asia
Larix	*Dasyscypha willkommii*	Canker	Eur.
	Helicobasidium mompa	Root rot	Asia
	Mycosphaerella larici-leptolepis.	Needle cast.	Do.
	Physalospora laricina	Shoot blight.	Do.
	Poria weirii	Root rot	N.A.
Picea	*Arceuthobium pusillum*	Witches'-broom.	Do.
	Chrysomyxa arctostaphyli	---do---	Do.
	C. deformans	Needle rust.	Asia
	Helicobasidium mompa	Root rot	Do.
	Poria weirii	---do---	Asia, N.A.
	Scleroderris lagerbergii	Canker	Eur.
Pinus	*Arceuthobium americanum*	Witches'-broom.	N.A.
	A. campylopodum	---do---	Do.
	Atropellis piniphila	Canker	Do.
	Cenangium ferruginosum	Dieback	Eur.
	Cercospora pini-densiflorae	Needle blight.	Asia
	Clitocybe tabescens	Root rot	N.A.
	Cronartium comandrae	Canker	Do.
	C. flaccidum	---do---	Eur., Asia
	C. fusiforme	---do---	N.A.
	C. himalayense	---do---	Asia
	C. quercuum	Gall	N.A.
	Elytroderma deformans	Needle cast	Do.
	Helicobasidium mompa	Root rot	Asia
	Hypoderma lethale	Needle cast.	N.A.
	Hypodermella sulcigena	---do---	Eur.
	Melampsora pinitorqua	Canker	Eur., N.A.
	Peridermium harknessii	Gall	N.A.
	P. stalactiforme	Canker	Do.
	Phacidium infestans	Needle blight.	Asia, Eur., N.A.
	Phomopsis pseudotsugae	Canker	Eur.
	Phymatotrichum omnivorum	Root rot	N.A.
	Poria weirii	--do---	Do.
	Scirrhia acicola	Needle blight.	Do.
	Scleroderris lagerbergii	Canker	Eur.
Pseudotsuga	*Arceuthobium douglasii*	Witches'-broom.	N.A.
	Phomopsis pseudotsugae	Canker	Eur.
	Poria weirii	Root rot	N.A.
Sequoia	*Phomopsis pseudotsugae*	Canker	Eur.
Tsuga	*Arceuthobium campylopodum.*	Witches'-broom.	N.A.
	Phomopsis pseudotsugae	Canker	Eur.
	Poria weirii	Root rot	Asia, N.A.
Platanaceae:			
Platanus	*Ceratocystis fimbriata*	Canker	N.A.
	Helicobasidium mompa	Root rot	Asia.
	Phymatotrichum omnivorum.	--do---	N.A.

Host, family, and genus	Pathogen	Symptoms	Continents
Ranunculaceae:			
Paeonia	*Cronartium flaccidum*	Leaf rust	Eur., Asia.
Rosaceae:			
Crataegus	*Clitocybe tabescens*	Root rot	N.A.
Malus	----do	----do	Do.
	Helicobasidium mompa	----do	Asia
Prunus	*Clitocybe tabescens*	----do	N.A.
	Helicobasidium mompa	----do	Asia
	Phymatotrichum omnivorum	----do	N.A.
Pyrus	*Clitocybe tabescens*	----do	Do.
Salicaceae:			
Populus	*Chrondoplea populea*	Canker	Eur., N.A., S.A.
	Helicobasidium mompa	Root rot	Asia
	Hypoxylon pruinatum	Canker	N.A.
	Melampsora allii-populina	Leaf rust	Eur.
	M. pinitorqua	----do	Eur., N.A.
	Phymatotrichum omnivorum	Root rot	N.A.
	Septoria musiva	Canker	N.A., S.A.
	Septotinia populiperda	Leaf blotch	Asia, Eur., N.A.
	Venturia populina	Leaf spot	Eur.
	V. tremulae	----do	Do.
Salix	*Helicobasidium mompa*	Root rot	Asia
Santalaceae:			
Comandra	*Cronartium comandrae*	Leaf rust	N.A.
Santalum	Sandal virus	Spike disease.	Asia.
Sapindaceae:			
Sapindus	*Phymatotrichum omnivorum.*	Root rot	N.A.
Scrophulariaceae:			
Castilleja	*Peridermium harknessii*	Leaf rust	Do.
	P. stalactiforme	--do	Do.
Euphrasia	*Cronartium flaccidum*	--do	Eur., Asia.
Melampyrum	----do	--do	Do.
	Peridermium stalactiforme	--do	N.A.
Nemesia	*Cronartium flaccidum*	--do	Eur., Asia.
Paulownia	Paulownia virus	Witches'-broom.	Asia.
Pedicularis	*Cronartium flaccidum*	Leaf rust	Eur., Asia.
Simarubaceae:			
Ailanthus	*Phymatotrichum omnivorum.*	Root rot	N.A.
Solanaceae:			
Schizanthus	*Cronartium flaccidum*	Leaf rust	Eur., Asia.
Tamaricaceae:			
Tamarix	*Phymatotrichum omnivorum.*	Root rot	N.A.
Taxodiaceae:			
Cryptomeria	*Cercospora cryptomeriae*	Canker	Asia
	Helicobasidium mompa	Root rot	Do.
Sequoia	*Cercospora cryptomeriae*	Canker	Do.
Tiliaceae:			
Tilia	*Strumella coryneoidea*	----do	N.A.
Tropaeolaceae:			
Tropaeolum	*Cronartium flaccidum*	Leaf rust	Eur., Asia
Ulmaceae:			
Celtis	*Helicobasidium mompa*	Root rot	Asia
	Phymatotrichum omnivorum	----do	N.A.
Ulmus	*Helicobasidium mompa*	----do	Asia
	Morsus ulmi	Virus wilt	N.A.
	Phymatotrichum omnivorum	Root rot	Do.
Verbenaceae:			
Tectona	*Olivea tectonae*	Leaf rust	Asia
Verbena	*Cronartium flaccidum*	----do	Eur., Asia

DANGEROUS FOREST DISEASES IN JAPAN

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Witches'-Broom of Paulownia

Virus (no specific name). In the case of normal paulownia, a bud sprouts in spring and grows to a new stem or a new branch, which bears about 10 pairs of opposite leaves. Stem growth ceases before the beginning of September. The axillary buds of the new stem or the branch do not sprout for this season, so that they usually have no lateral shoots for the current season. In the diseased paulownia, on the contrary, a bud sprouts in spring and grows to a new stem or to a new branch which does not cease its growth until late in autumn and the primary axillary buds sprout immediately. The sprouting of the axillary buds and the growth of the shoots and branches without any restriction are the causes of the symptom of the witches'-broom. The branches and shoots of the diseased tree are slender and brittle and show an extreme negative geotropism. The color of diseased branches and shoots becomes yellowish green.

There are two sorts of leaf-forms in healthy paulownia. The one is the leaf-form seen in young trees 1 or 2 years old and the other is that seen in older trees. The former show both large and small incisions. With the growth of the tree, the leaves which have only large incisions increase in number, and within 2 or 3 years the whole tree comes to have leaves with large incisions on the margin. In the diseased tree, however, the leaves with both large and small incisions appear even though it becomes more than 2 years old. Usually all the leaves on the diseased shoots are abnormally thin and narrow and are uneven on the surface. Their color is yellowish. Malformed leaves are often observed on the diseased shoots.

This is the most serious disease of paulownia trees in Japan, and causes severe damage in many plantations in the central and southern parts of this country. Heavily infected trees are stunted and killed. Insect transmission of this disease is not known.

Intercontinental spread is possible through shipment of infected trees or rootings. Importation of living plant material other than pollen or seed should be forbidden.

Distribution: In Japan it is common from the central to southern part and scattered in the northern parts except Hokkaido. Also found in China; Korea (?).

Hosts: Scrophulariaceae—
Paulownia tomentosa,
P. fortunei,
P. kawakamii.

Literature:

Tokushige, Y. Witches' broom of *Paulownia tomentosa* L. Jour. Fac. Agr., Kyushu Univ. 10: 45–67. 1951.

——— Witches' broom of *Paulownia tomentosa* L. On the occurrence of the witches' broom affected trees in paulownia forest (Japanese with English resume). Jour. Jap. Forest Soc. 34: 4–7. 1952.

Bacterial Blight of Chestnut

Pseudomonas castaneae (Kawamura) Savulescu. The disease is most conspicuous in buds and young shoots but also occurs on the leaves, veins, petioles, and bracteal leaves, the last being usually attacked first. The sign of the disease in the early stage is water-soaked spots on the leaves and young shoots in which the cortical parenchyma is destroyed, forming bacterial cavities and resulting in brown cracks. Leaves attacked when young become distorted and leaves of infected buds shrivel and die. White and yellow bacteria are isolated from the diseased lesions, of which the former is pathogenic to chestnut and found associated with the latter.

A short rod, 1.0–1.8μ × 0.8–1.2μ, motile, with one to five polar flagella; singly or in pairs, no spores, no capsules, Gram-negative, facultative anaerobic, beef agar colonies white, round, slightly undulate and viscid, bouillon clouds without pellicle; potato decoction agar colonies white, radiately rugose, gelatin not liquefied, diastatic action absent, milk peptonized without coagulation, indol and hydrogen sulphide not produced, nitrate and methylene blue reduced, acid from dextrose, saccharose and glycerine without gas, neither acid nor gas produced from lactose, no gas from maltose and potassium.

The distribution of this bacterium is limited to the southern districts of Japan. Intercontinental spread may be possible only by infected living trees, though pollen dissemination is also possible.

Range: Japan-Kyushu and Chugoku Districts.

Host: Fagaceae—*Castanea crenata*,
C. mollissima.

Literature:

Kawamura, E. Bacterial blight of chestnut (Japanese with English resume). Ann. Phytopath. Soc. Jap. 3: 15–21. 1934.

Needle Cast of Larch

Mycosphaerella larici-leptolepis K. Ito & K. Sato. The earliest indication of the disease usually occurs during the first week in July. Generally, scattered spots on the needle are first infected. They are at first minute, brown, and surrounded by a faint yellow halo. As the disease progresses, these spots increase in size and coalesce to attain a width of 1 mm. or more. Lesions are present 5 to 7, rarely 20 per needle. The discolored needles bear small black fruit spermogonia on the upper surface of the dead area. From a distance the infected trees give the appearance of having been scorched by fire or injured by late frost.

Spermogonia, 83–165 × 74–143μ, are formed on both green and fallen needles throughout autumn and winter. The mature spermogonia are filled with a great number of hyaline, rod-shaped spermatia, 3–5 × 0.5–1μ in size.

Perithecia are produced on the fallen needles in contact with the soil. Early in March structures that are interpreted to be perithecial initials are observed. Perithecia develop either singly or in groups, at first embedded within the host tissue, but later becoming erumpent. Ascospores mature in May to July. Mature perithecia are amphigenous, single or in groups, partially erumpent, globose, 88–157 × 84–142μ. Asci are clavate-cylindrical, 49–99 × 7–12μ, contain 8 ascospores. Paraphyses are absent. Ascospores are hyaline, unequally two-celled, constricted at septum, 11–18 × 3–5μ.

This is one of the most important diseases of larch forests. The characteristic symptom of this disease is a browning of the needles over all or part of the crown. This browning is most conspicuous in summer and autumn and gives the impression that the tree is dying. Later the diseased needles drop off, leaving the trees with all or portions of their crowns thin and the remaining needles confined to tufts at the end of the branches. Repeated serious defoliations bring about a considerable decrease in growth increment.

The source of infection is diseased needles, and intercontinental spread may be possible by seedlings and stocks bearing needles.

Range: Northern and central parts of Japan.

Hosts: Pinaceae (species listed in order of susceptibility)—
Larix decidua (Planted in Japan)
L. gmelini var. *olgenis* (Planted in Japan)
L. gmelini var. *japonica*
L. leptolepis (*L. kaempheri*)

Literature: Ito, K.; Sato K.; and Ota, N. Studies on the needle cast of Japanese larch—I. Life history of the causal fungus, Mycosphaerella laricileptolepis sp. nov. Bul. Govt. Forest Expt. Sta. 96 :69–88. 1957.

Shoot Blight of Larch

Physalospora laricina Sawada. Stems of seedlings and shoots of young trees are attacked. Lesions are more or less sunken and frequently exude resin. Diseased stems and twigs are girdled, killed, and then defoliated. Many dieback twigs are seen in the crowns of heavily diseased trees. Perithecia are produced under the epidermal tissues of killed twigs and stems.

Perithecia are black, globose, erumpent, 368μ in diameter. Asci are clavate, rounded at apical portion, hyaline, 114–135 × 22–26μ. Paraphyses are 3μ in diameter, rarely branched. Ascospores are elliptic, smooth, 24–27 × 13μ. Imperfect stage, *Macrophoma*, is found on both twigs and leaves.

This is currently the most serious disease of larch forests in Japan. Though trees of all ages are susceptible, young trees are very heavily attacked. Infected trees are malformed and growth is greatly reduced.

International spread may be possible by infected trees or scions. Importation of living plant material other than pollen or seed should be forbidden except for experimental use following rigorous inspec-

tion and retention in quarantine until all latent infections have had time to appear.

Range: In Japan it is common from Hokkaido south to the Tohoku District.

Hosts: Pinaceae (species listed in order of susceptibility)—
Larix decidua (Planted in Japan)
L. gmelini var. *olgenis* (Planted in Japan)
L. gmelini var. *japonica*
L. leptolepis

Literature:

Sawada, K. Fungi inhabiting conifers in the Tohoku District II. Fungi on various conifers except "Sugi". (Japanese with Latin Diagnoses) Bul. Govt. Forest Expt. Sta. 46: 111–150. 1950.

Uozumi, T. Shoot blight of larch in Hokkaido (Japanese). Forest Protect. News (Tokyo), 7: 156–158. 1958.

Anthracnose of Acacia

Glomerella acaciae (K. Ito & Shibukawa) K. Ito (syn. *Physalospora acaciae* K. Ito & Shibukawa). Symptoms of the disease first appear on the plant as punctate brown lesions, which later enlarge and attain 5–10 mm. in diameter and become dark brown in color. The disease attacks all of the above ground parts of *Acacia* seedlings including leaves, stems, petioles, and branchlets. During wet periods the lesions elongate, coalesce, and very frequently girdle entire stems and petioles, causing a rapid wilting, early defoliation, and subsequent death of the shoot. The fungous invasion of the young succulent shoot is especially rapid and severe. A number of the affected seedlings have dead tops with a few basal living branchlets. Under moist conditions, conidial masses of salmon pink color are abundantly produced on the lesions. About the end of October small dark brown to black perithecia are irregularly scattered on the surface of the dead area.

Acervuli erumpent, scattered or gregarious, 100–140μ in diameter, conidiophores hyaline, cylindrical or fusoid, 6–15 × 2–3μ, setae among conidiophores, 1- or 2-celled, few or numerous, dark brown tapering at the apex 24–72 × 3–6μ, conidia hyaline, straight with round ends, 12–18 × 4–6μ, 1-celled. Perithecia single or in groups, partially erumpent, globose, slightly papillate, 54–141 × 60–114μ, asci ovate-oblong with a collar extending into the apical wall, 8-spored, 36–60 × 6–9μ, paraphyses broad in width, acute in apical portion, 39–55 × 3–8μ, ascospores hyaline, ovate or elliptical, arranged irregularly, 1-celled, 10–15 × 3–6μ.

This is the most serious disease of *Acacia* seedlings (especially *A. dealbata*) in Japan. Losses are very heavy, more than 90 percent mortality in some nurseries.

One of the most important features of this disease is the penetration of the pathogen into the seed, where it may pass the winter as a dormant mycelium. The fungus is usually detected in about 5 percent of the seeds collected in Kyushu. The infected seeds are thus the most important sources of the primary infections, which appear in the nurseries. It has been thought that the causal organism might be imported from abroad with seeds into Japan. In importation of *Acacia*

seeds, seed treatment with disinfectants or hot water bath is necessary for anthracnose-free countries.

Range: Central and southern districts of Japan.

Hosts: Leguminosae—
Acacia dealbata
A. mollissima

Literature: Ito, K., and Shibukawa, K. Studies on some anthracnoses of woody plants—III. A new anthracnose of Acacia with special reference to the life history of the causal fungus. Bul. Govt. Forest Expt. Sta., 92 : 51–64. 1956.

Leaf Blotch of Poplar

Septotinia populiperda Waterman & Cash. Symptoms first appear in early spring as small brown spots on the young leaves, frequently at or near the margin, but also at any point on the leaf blade. The spots usually increase rapidly in size, soon become gray at the center, and have an irregular but sharply defined margin. On the most susceptible species and hybrids, large areas of the leaf blade become invaded by the fungus.

On the lower surface, the leaf blade and particularly the veins in the affected area become dark brown with white sporodochia. On the upper surface, small white masses of conidia appear, usually developing in concentric circles. Along the margin of the blotch, white mycelial fans are frequently formed just below the cuticle of the upper leaf surface. Two or more of the spots may coalesce, resulting in almost complete invasion of the leaf tissue. Early defoliation usually follows, and the young shoots of the most susceptible poplars may thus be entirely defoliated by late summer. On the fallen diseased leaves on the ground, thin, small, black sclerotia of the fungus are abundantly produced in late October. For morphological characteristics of the pathogen, see Waterman and Cash (1950).

In 1956, the first collection of this disease in Japan was made in Tokyo. Since that time, further surveys showed a more extensive distribution of the disease. Now, the fungus is distributed widely throughout Japan almost everywhere poplars are cultivated. Leaf blotch is the most important disease of poplar leaves and causes severe damage. Japanese pathologists believe that the pathogen was probably imported with cuttings from Europe or America to Japan after World War II.

International spread is chiefly by cuttings, and imported cuttings must be surface sterilized before planting.

Distribution: Japan, North America, Europe.

Host: Salicaceae.

Susceptible: *Populus simonii*, *P. koreana*, *P. davidiana* × *P. canescens*, *P. tremula* × *P. tremuloides*, *P. canadensis*, *P. serotina*, *P. regenerata*, *P. marilandica*, "Rochester" poplar, *P. euramericana*, I–455, I–214, I–154, I–293, LK–79, LW–42, "Wettstein" poplar, "Jacometii" poplar, *P. japonogigas*, *P. eugenii*, *P. berolinensis*. (All planted in Japan).

Very susceptible: *P. nigra* ×*maximowiczii* (Kamabuchi-1,2), *P. charkowiensis* ×*trichocarpa*, *P. robusta*, *P. gelrica*, "Leipzig" poplar, *P. nigra* ×*laurifolia* (All planted in Japan).

Literature:

Waterman, Alma M., and Cash, Edith K. Leaf blotch of poplar caused by a new species of Septotinia. Mycologia 42:374–384. 1950.

Ito, K. Parasitic diseases of poplars in Japan. Forestry Agency of Japan, 22 pp. 1959.

Violet Root Rot

Helicobasidium mompa Tanaka. Slender roots attacked by the fungus become yellowish-brown or blackish-brown in color. They are softened, rotted, and finally disappear. In the case of severe damage, the cambial portion of large roots is heavily destroyed, only the woody tissue and the bark with no trace of small roots remain. On heavily affected roots, a great number of sclerotia buried in the cork layer are seen very frequently. Purple rhizomorphs creep up the surface of the roots and the trunk, increasing in diameter towards the ends. When the roots are severely injured it is not rare for numerous small roots to develop. Diseased plants may thus escape death at least temporarily.

Soon after the aggregation of rhizomorphs reach the basal portion of the host, they develop into a mycelial mat or a sporophore. The sporophore formed on the basal part of the tree spreads upwards as well as sidewards covering the surface of the trunk. During May to July, the formation of the hymenial layer causes the surface of the sporophore to become powdery white. Many of the diseased trees at first show no remarkable changes in appearance above the ground, but, by and by, some of them are noticeably weakened and finally killed. In heavily affected trees, the leaves become more or less smaller, yellowish, and fall earlier than in the healthy ones. In extreme cases the entire subterranean portion of the diseased plant is almost completely destroyed by secondary invasion of wood-rotting fungi.

Sporophores develop around the basal portion of the trunk up to a height of 10 cm. or more, sometimes leaving here and there narrow parts uncovered. They are sessile, resupinate, often irregularly lobed, velvety, and membranaceous. Surface of the fruit-body, which was deep purplish brown in early spring, becomes whitish or light pink in color during the later part of the same season. In spore-bearing sporophores, four or five layers are anatomically distinguished. Basidia do not arise from preformed resting cells, probasidia. Young basidia are hyaline, smooth, erect, club-shaped; mature basidia are curved, generally 3-septate, 30–50 × 5–10μ, the 4 sterigmata are elongated, narrowed towards end, 10–15μ long, tetraspored. Basidiospores are hyaline, ovoid, slightly curved, 12–25 × 5–9μ in size.

This is one of the most important soil-borne diseases in Japan. Almost all tree species are susceptible. Spread of this disease is mainly by translocation of infected seedlings or stocks. Many different control practices have been tried and recommended at various times, but most of them have proved either ineffective or only partially successful.

Distribution: Japan, Korea, and Formosa.

Hosts: Over 100 species, both ligneous and herbaceous, belonging to various genera are known as the hosts of the fungus. Some woody hosts containing introduced species are listed as follows:

Ginkgoaceae—*Ginkgo biloba* (very susceptible)
Pinaceae—
Picea excelsa (Planted in Japan)
Larix leptolepis
Pinus parviflora, *P. densiflora* (very susceptible)
P. strobus (very susceptible) (Planted in Japan)
P. taeda (very susceptible) (Planted in Japan)
Taxodiaceae—*Cryptomeria japonica*
Cupressaceae—*Thuja occidentalis*, *Chamaecyparis obtusa*
Salicaceae—
Populus maximowiczii, *P. nigra* (Planted in Japan)
P. nigra × *P. maximowiczii* ("Kamabuchi"–1) (very susceptible), *Salix bakko*
Juglandaceae—
Juglans sieboldiana (very susceptible)
Platycarya strobilacea
Fagaceae—
Castanea crenata (very susceptible)
C. mollissima (very susceptible) (Planted in Japan)
Quercus acutissima
Ulmaceae—
Ulmus pumila
Celtis sinensis
Moraceae—
Broussonetia kazinoki (very susceptible)
Ficus carica
F. elastica
Morus alba (very susceptible)
Magnoliaceae—*Liriodendron tulipifera* (very susceptible) (Planted in Japan)
Platanaceae—*Platanus orientalis*
Rosaceae—
Malus pumila (very susceptible)
Prunus serrata (very susceptible)
Leguminosae—
Cercis canadensis (Planted in Japan)
Robinia pseudoacacia (very susceptible) (Planted in Japan)
Anacardiaceae—*Rhus verniciflua* (very susceptible)
Aceraceae—*Acer campestris* (Planted in Japan)

Literature:

Ito, K. Studies on "Murasaki-monpa" disease caused by Helicobasidium mompa Tanaka. Bul. Govt. Forest Expt. Sta. 43: 1–126. 1949.

Suzuka, N., et al. Studies on the violet root rot of sweet potatoes. (Japanese with English resumé.) Bul. Natl. Inst. Agr. Sci. Sr. C. 8:1–173. 1957.

Needle Blight and Canker of Cryptomeria

Cercospora cryptomeriae Shirai. First symptoms on seedlings are brownish discoloration in needles and stems. Infected needles and twigs become dark brown and die. Under favorable moisture conditions, needle and twig blight progresses rapidly and causes the death of seedlings. Young, vigorously growing shoots and stems become infected around the base of a diseased needle or small twig.

The affected area becomes blackened and slightly depressed. As the tissue dries, it turns dark brown. Stems less than 10 mm. in diameter may be girdled by the fungus in one growing season.

Dark greenish fruit-bodies of the causal fungus are produced abundantly on the diseased needles and cankers of seedlings. As cankers increase in size, the cambial tissue may be killed inward, producing a flat-faced canker, swollen at the sides, and distorting the stem. These cankers usually are perennial and increase longitudinally for many years. Frequently the rapid growth of the stem tissue prevents the girdling of the stem, and trees may live for many years with one or more disfiguring but non-girdling cankers.

Stromata dark brown, semi-spherical, fascicles dense, conidiophores light yellowish-brown, rarely septate, non-branched, 4–6 × 40–80μ, conidia pale yellowish-olivaceous, obclavate to obclavate-cylindric, straight or slightly curved, echinulate, 3 to 7 septate, 5–9 × 30–85μ.

This is the most important disease of conifers in Japan. Seedlings and young trees are very susceptible. Infected seedlings are killed by blighting and canker. Losses are very heavy, up to 90 percent mortality in many nurseries. Intercontinental spread is possible through shipment of infected trees or cuttings. The causal fungus is very similar to *Cercospora sequoiae* Ellis & Ev. described on *Sequoia gigantea* in the United States of America. Spraying with fungicides in the nursery is very effective for control of the disease.

Distribution: Japan, Formosa.

Hosts: Taxodiaceae—

Cryptomeria japonica
Sequoia gigantea (Planted in Japan)

Literature:

Ito, K. The cause of canker of Cryptomeria japonica (Japanese). Plant Protect. (Tokyo) 6: 176–179. 1953.

———, Shibukawa, K., and Kobayashi, T. Etiological and pathological studies on the needle blight of Cryptomeria japonica I. Morphology and pathogenicity of the fungi inhabiting the blighted needles. (Japanese with English resume.) Bul. Govt. Forest Expt. Sta. 52: 79–152. 1952.

———, Shibukawa, K., and Kobayashi, T. Blight of Sequoia gigantea seedlings caused by Cercospora cryptomeriae Shirai (Japanese). Jour. Jap. Forest Soc. 40: 407–410. 1958.

Needle Blight of Pine

Cercospora pini-densiflorae Hori & Nambu. Yellowish-brown to gray lesions appear mostly on the upper half of the needle as the disease progresses, especially on 1- to 3-year-old trees. The needles die to such an extent that it is known as "leaf-blight."

Dark brown stromata fill the stomatal openings or as large as 60μ in diameter, fascicles dense to very dense; conidiophores dark brown, rarely septate, not branched, sparingly geniculate, 2.5–5 × 10–45μ; conidia pale yellowish olivaceous, obclavate to obclavate-cylindric, straight to curved, 3–7 septate, rounded to obconically truncate base, obtuse tip, 3–6 × 20–60μ.

This is the most serious disease of pine seedlings. Heavily infected seedlings die. Losses are heavy, up to 80 percent mortality in some nurseries.

International spread is possible only through shipment of infected seedlings. Controls by spraying with Bordeaux mixture or other fungicides are successful.

Distribution: Japan: Kyushu, Shikoku. Formosa: Shizuoka.
Hosts: Pinaceae.
Susceptible: *Pinus densiflora*, *P. thunbergii*, *P. massoniana* (Planted in Japan), *P. luchuensis.*
Very susceptible: *P. radiata*, *P. strobus*, *P. pinaster*, *P. canariensis* (All planted in Japan).

Literature:

Chupp, C. A monograph of the fungus genus Cercospora. Ithaca, New York. 440 pp. 1953.
Nukumizu, T. Needle blight of pine (Japanese). Forest Protect. News (Tokyo) 5:264. 1956.
Ito, K. Diseases of Pinus strobus in Japan. (Japanese). *Ibid.*, 10:43–46. 1961.

The following five diseases are listed but not described. Detailed information on them will be supplied upon request to the authors.

Virus Disease of Chestnut

Virus (No specific name)
Distribution: Japan (Ibaragi and Yamagata Prefectures).
Hosts: *Castanea crenata*, *C. mollissima.*

Gall Rust of Pine

Cronartium quercuum Miyabe
Distribution: Japan, Formosa, Korea.
Hosts: *Pinus densiflora*, *P. thunbergii*, *P. sylvestris* (Planted in Japan).
Alternate hosts: *Quercus*, *Castanea*, *Cyclobalanopsis*, *Shiia.*

Needle Rust of 5-Needle Pine

Coleosporium paederiae Diet.
Distribution: Japan, Formosa.
Hosts: *Pinus strobus* (Planted in Japan).
Alternate host: *Paederia chinensis.*

Brown Spot of Tung

Cercospora aleuritidis Miyake syn. *Mycosphaerella aleuritidis* (Miyake) Ou.
Distribution: Japan, China, etc.
Host: *Aleurites fordii.*

Needle Blight of Redwood

Cercospora exosporioides Bubak.
Distribution: Japan (Kyushu and Tokyo), etc.
Hosts: *Sequoia sempervirens* (Planted in Japan). *Larix leptolepis.*

DANGEROUS FOREST DISEASES IN INDIA

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Spike Disease of Sandal

Virus (No specific name). A serious virus disease of sandal (*Santalum album* L.) in south India. The symptoms first appear in one or more branches, in which new leaves become progressively reduced in size, pale and stand stiff and crowded on shortened internodes, giving a spikelike appearance. Spike eventually spreads over the entire tree which exhibits continuous growth. In advanced stages of disease, flowers and fruits do not develop. If a branch is spiked after inflorescence is formed, fruits become reduced in size, or flowers exhibit phyllody. Haustoria and fine root ends die. In spiked branches and leaves, starch is significantly more, while nitrogen and ash less than in normal leaves. The spiked tree never recovers and dies in a few years.

Disease is transmitted by grafting and not communicable by sap transmission. Intracellular cell inclusions are demonstrated. The claim that *Jassus indicus* is a vector for the disease is not confirmed and any other insect is yet unknown. Several plants show symptoms of spike and some of them serve as hosts for sandal.

Eradication of diseased trees or lopping spiked branches does not reduce incidence of disease. Marking out susceptible and resistant hosts, and propagating sandal on the latter is suggested. However, some principal susceptible hosts like *Lantana camara*, are excellent nurse crops for sandal and their eradication is practicable. Natural selection of resistant trees and propagating them through seeds has not controlled the disease. In nature, sandal grows healthy, free from spike under shade. Control of spike may be attempted by raising sandal under a light canopy of trees to serve as good sandal hosts and at the same time afford some degree of resistance to spike.

The disease has killed sandal in large tracts in south India. It spreads in the direction of wind. Although sandal is now naturalized in India, it is suggested that it was introduced from Java in the remote past. The disease may have occurred naturally on some native plants, where it is harmless, and from which it transferred to sandal, finding the exotic highly susceptible.

The disease is known only in India. Not reported from Malaysian islands and Australia where sandal occurs naturally. Export of living sandal from India except seeds for experiment should be forbidden.

Hosts: Santalaceae—*Santalum album* L.

Literature: Coleman, L. C. Spike disease of sandal. Mycol. Ser., Bul. 3, Dept. Agr., Mysore State. 1917.

Deodar Witches'-Broom

Peridermium cedri (Barclay) Sacc. A needle rust of deodar (*Cedrus deodara* Loudon). Attacks young needles of the current

year's shoots. Needles become short and curved and shed earlier than normal, so that foliage is thin. Affected branches and, rarely, the entire tree is transformed into witches'-brooms. Causes large-scale mortality, particularly in young trees, or death of affected branches in large trees.

The rust is known only as pycnia and aecia on deodar. Pycnia abundant, superficial, 0.08 mm. broad, 0.04 mm. deep. Aecia 10 to 12 on each needle on upper surface, in one row on one or the other side of the middle line, but at distal end in two rows. Aecia are superficial, up to 0.5 mm. broad and 0.16 mm. deep. Aeciospores in chains, yellow with orange-yellow granular contents, round or oval, 15–21 × 14–17μ. The epispore is thick and striated. The aeciospores ripen in May–June. Pseudoperidium is delicate, 1-cell thick, and falls apart soon.

This is a serious disease of deodar in the western Himalayas in India, particularly under wet conditions. Overwintering of aeciospores does not occur. The fungus overwinters as mycelium in the needles and in the shoots containing them, to infect new needles in the next season.

Systematic removal of diseased plants or affected branches has been found to bring the disease under control.

Deodar is grown as an exotic in America and elsewhere. Importation of living plant material other than seed should be forbidden in foreign lands.

Hosts: Pinaceae—*Cedrus deodara* Loud.

Literature: Troup, R. S. *Peridermium cedri* as a destructive fungus. Indian Forester 38:222–223. 1912. *Ibid.*, 40:469–472. 1914.

Chir Pine Blister Rust

Cronartium himalayense Bagchee. A stem rust of chir pine (*Pinus roxburghii* Sargent), a 3-needle pine in west Himalayas (India). The symptoms include resin bleeding, cankers, and cracking bark. Plants become dwarfed with poorly developed needles which shed early. Pycnia of pinhead size appear through the bark and exude orange-yellow fluid. Later, abundant aecial cups burst through the ruptured bark as orange-yellow blisters.

Cronartium himalayense is a heteroecious rust. Pycnia and aecia occur on chir pine; and uredia, telia, and basidia on *Swertia elata*, *S. angustifolia*, and *S. cordata*. Pycnospores are hyaline, oval, 3–5 × 1.5–2.3μ. Aecia are cylindric, 4 to 12 mm. long, 2 to 6 mm. broad, with a persistent peridium which ruptures irregularly at the sides when mature. Aeciospores are orange yellow, oval, 22–31 × 13–19μ with a thick, verrucose wall, and develop in chains. They infect swertia situated within 2 to 3 miles.

Orange-yellow uredia appear on leaves and stems of swertia and are subepidermal. Uredospores are orange yellow, oval, thick-walled, spiny, about 22 × 16μ. They reinfect swertia. Telia develop later in the uredia or form separately. Teliospores are columnar, hairlike, up to 3 mm. long and 0.1 mm. broad, germinate in situ into 4-celled promycelium, each cell developing a sporidium, borne on a sterigma. Sporidia are hyaline, globose, 5.5 to 6.5μ broad. They infect pines occurring within a range of 200 yards.

The disease causes mortality particularly to young plants in plantations and less so in natural forests. Older plants are less susceptible

to the disease, the attack occurring mainly on branches which are killed by girdling.

Control of the disease may be possible by eradication of the annual weed swertia from the affected chir pine forests and up to a distance of 200 yards from them. Removal of diseased chir and raising broad-leaf species with chir to serve as windbreaks to prevent the ingress of sporidia into pine forests is suggested for control. Since a plantation crop, at young age, is particularly susceptible, "Selection System" is advisable.

An indigenous species to India only, chir is raised in south and east Africa. Importation of living plant material except seeds should be forbidden except for experimental purposes following rigorous inspection and retention in quarantine until all latent infections have had time to appear.

Hosts: Pinaceae—*Pinus roxburghii* Sarg.

Literature: Bagchee, K. Investigations on the infestations of *Peridermium himalayense* Bagchee on *Pinus longifolia*. II. *Cronartium himalayense* n. sp. on *Swertia* spp. Indian Forest Rec. (Bot. Ser.) 18:66. 1933.

Spruce Rust

Chrysomyxa deformans (Diet.) Jaczew. A microcyclic rust on *Picea smithiana* (Wall.) Boiss. Only the current year's shoots are attacked and become deformed. All needles in the shoot are infected, become stunted, small, thick, twisted with apices curved. When young, emits a disagreeable odor. The short affected orange-yellow tassels stand out prominently. Orange-red telia occupy two continuous flattened beds on the upper surface and two rows of smaller ones below. Teliospores are yellow, smooth, round, 9.5 to 12μ broad.

Attacks mature trees and is widespread.

This Himalayan spruce is now raised in Europe and America. Care should be taken to see that the rust does not appear in these new homes.

Hosts: Pinaceae—*Picea smithiana* (Wall.) Boiss.

Literature: Butler, E. J. Some Indian forest fungi. III. Indian Forester 31:611–617. 1905.

Teak Leaf Rust

Olivea tectonae (Racib.) Thirum. A microcyclic rust on teak (*Tectona grandis* L.), attacking leaves only, the lower surfaces of which turn orange yellow due to formation of sori. Upper surface of leaves may present a gray appearance due to the formation of flecks which correspond to the position of sori below.

Uredia almost plaster the lower surface, minute, subepidermal, orange yellow, pulverulent, 0.2 to 0.5 mm. in diameter; uredospores orange yellow, globose to ellipsoid, densely echinulate, 20–27 × 16–22μ; paraphyses marginal, cylindric, incurved, coalescent at the base with orange-yellow contents, wall up to 2.5μ thick. Telia develop within uredia or separately, waxy, orange yellow, paraphyses same as in uredia. Teliospores clavate, sessile, borne in clusters on basal cells with orange-yellow contents, 38–51 × 6–9μ; teliospores germinate within the

sorus at maturity, promycelium external, four-celled; basidiospores round to elliptical.

It is widespread in teak forests in India. Damage is severe in young plantations, especially in nurseries where young plants are retarded in growth due to premature defoliation. Opening the canopy is helpful in controlling the disease.

Teak is indigenous in India, east Pakistan, Burma, Siam, and Java. It is raised as an exotic in Central America, Sudan, and elsewhere. Importation of living plant material other than seeds should be forbidden.

Hosts: Verbenaceae—*Tectona grandis* L.

Literature: Thirumalachar, M. J. Telia of the leaf-rust of teak. Current Science 18: 175–177. 1949.

Shisham Wilt

Fusarium solani (Martius) Appel and Wollenw., forma *dalbergiae* Gordon. A fungus causing wilt of shisham (*Dalbergia sissoo* Roxb.) in north India and Pakistan. The disease is systemic. Early symptoms are drooping leaves and leaflets which lose the normal green color, gradually turn yellow, become dry and are shed early, rendering the branches increasingly bare. Diseased trees die within 4 to 6 months. The affected roots gradually become devoid of finer branches and rootlets, on which nodules are scarce or absent. The outer living wood of roots shows a pink stain which may progress up the stem a short distance in advanced stages of the disease. The pathogenicity of the fungus is established by inoculation of healthy plants in which symptoms are reproduced.

Fusarium solani forma *dalbergiae* is a soil inhabiting fungus. Infection occurs through roots. Trees of all ages are attacked though seedlings and saplings are less susceptible. A definite correlation exists between soil texture and incidence of the disease. The disease is absent in shisham occurring naturally on sandy soils such as river beds, where the species is one of the early colonizers. The disease is also rare in plantations where soil is loose and sandy. In natural forests and also in plantations where the soil is stiff and clayey, the disease incidence is high. Freedom from the disease can thus be secured if shisham plantations are raised in soils containing a high proportion of sand and less of silt and clay. Irrigation with good soil drainage is known to maintain healthy stands.

A species indigenous to India and west Pakistan and that is being tried as an exotic in the Middle East countries like Sudan, where care should be taken to see that the disease does not appear.

Hosts: Leguminosae—*Dalbergia sissoo* Roxb.

Literature: Bakshi, B. K., et al. Wilt disease of shisham. I. Indian Forester 80: 316–322. 1954; II. *Ibid.* 81: 276–281. 1955: IV. *Ibid.* 83: 505–511. 1957; V. *Ibid.* 83: 555–558. 1957.

Casuarina Wilt

Trichosporium vesiculosum Butler. A stem and root parasite of *Casuarina equisetifolia* Forst. in plantations in south India. Appears on *Casuarina* bark as blisters, which rupture and expose a thick

black powdery mass of spores. The spores are brown, subglobose to oval, 1-celled, wall darker, smooth, 4.9–6.1 × 2.7–3.7μ. They are windblown and infect trees through wounds caused by pruning or lopping of branches. From the stem, the fungus travels into the roots. Secondary spread of the disease occurs through infected roots by root contact and grafting. Owing to long spreading lateral roots, the disease spreads rapidly in plantations. Diseased trees occur in groups. Infected trees are killed in 6 to 8 months.

Prevention of pruning and lopping will eliminate or reduce primary infection. Secondary spread of the disease may be checked by trenching diseased trees. All dead trees including infected ones should be removed.

This is the most serious disease of *Casuarina* in plantations in south India. Trees are attacked mainly in plantations of pure *Casuarina* with usually 7 years rotation. Losses are high, up to 75 percent mortality in some stands.

Casuarina is indigenous in Australia, the Pacific Islands, and Malaya and is also planted in Central and North America. It was introduced into south India where it is now naturalized. Export of diseased plant material should be prevented.

Hosts: Casuarinaceae—*Casuarina equisetifolia* Forst.

Literature: Bakshi, B. K. Mortality of *Casuarina equisetifolia* Forst. Indian Forester 77 : 269–276. 1951.

Sal Root Rot

Polyporus shoreae Wakef. A common fungus in sal (*Shorea robusta* Gaertn.) forests of north India but becomes of economic significance in wet sal type only where annual rainfall is over 75 inches. The disease is encountered on good sites with sal in quality classes I, II, and III (over 100 ft., 80–100 ft., and 60–80 ft. height growth, respectively).

The fungus attacks healthy uninjured roots and progresses towards the collar but does not normally advance into the stem. Light yellow mycelial felts develop in patches on bark. White pocket rot develops in bark and sapwood. The affected tree shows symptoms of top dying after about three-fourths of the root system is attacked. Dying extends downwards till the tree becomes dead or, as commonly happens, the diseased tree becomes windblown due to decayed roots. Diseased trees occur in groups. Sporophores develop at the base of the tree. They are sessile, pileate, brown.

The disease is serious in wet sal forests of north Bengal and Assam. Healthy, vigorous trees of advanced age are attacked. Mortality may be 20 percent or more. High moisture in soil favors activity of the fungus. Control burning, recommended in silvicultural practice in wet sal forests, will reduce soil moisture and check growth of weeds which thereby reduces disease incidence. Dying and dead trees should be removed and converted, since timber quality is not affected.

Shorea robusta and other species of *Shorea* occur in India, Pakistan, Burma, and the Far East. The occurrence of *Polyporus shoreae* outside its known range in India should be determined.

Hosts: Dipterocarpaceae—*Shorea robusta* Gaertn.

Literature: Bakshi, B. K., and Boyce, J. S. *Polyporus shoreae* root rot of sal. Indian Forester 85 :656–658. 1959.

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Sal Top-Dying

Hypoxylon mediterraneum (de Not.) Mill. var. *microspora* Miller is a widespread fungus on sal (*Shorea robusta* Gaertn.) in north India. Becomes of economic significance only on poor sites, with sal in quality classes III and IV (60–80 ft. and below 60 ft. height growth, respectively). Top-dying of sal occurs on deteriorating sites. Trees thus weakened become attacked by *Hypoxylon*, which hastens death of trees or prevents their recovery. The fungus is parasitic and kills tissues on which fruit bodies develop so that they are found on bark of dead branches and stems of dying and dead trees. Stroma effused in wide patches formed within the bark, black, carbonaceous, 1 to 2 mm. thick; perithecia with ostiolar necks protruding beyond ectostroma and with circular depression around ostioles, cylindrical, black and carbonaceous, 1–1.6 × 0.15–0.25 mm.; asci cylindrical intermixed with paraphyses; ascospores ovoid to ellipsoid, dark brown to almost black, one guttulate, 8.5–12.2 × 7.1–9.2μ.

Since weakened sal trees on poor sites are attacked by the fungus, the vigor of the stand should be restored by improving site conditions through proper silvicultural treatment such as fire protection, closure to grazing, and contour trenching to minimize water runoff and thus increase soil moisture. Dead and dying trees should be removed early to permit salvage of timber which is sound, and also to reduce fire hazard from dead branches and bark falling on ground.

The pathogen is known from the Mediterranean area on oaks.

Hosts: Dipterocarpaceae—*Shorea robusta* Gaertn.

Literature: Boyce, J. S., and Bakshi, B. K. Dying of sal. Indian Forester 85: 585–588. 1959.

Khair Heart Rot

Fomes badius Berk. A perennial heart-rot fungus almost exclusively on khair (*Acacia catechu* Willd.) in both natural and artificial stands. Heartwood, valued for katha and cutch, becomes decayed. Rot is white and spongy. Sporophores develop commonly and serve to identify diseased trees, which otherwise grow apparently healthy.

Sporophores perennial, hard, woody, usually 7 × 4 × 3 cm.; upper surface brown to black, sometimes rimose with age; hymenial surface dull brown, pore tubes distinctly stratified, basidiospores yellowish-brown, 5.8–7.5 × 4.1–5.8μ, windblown, cause infection through wounds due to lopping for fuel, fodder, seed collection, etc., or animal damage.

Causes great economic loss to khair, an important cash crop tree. Attacks trees above 4 inches d.b.h. when heartwood develops.

Since *Fomes badius* is a wound parasite, control of the disease is secured by prevention of injury to trees. Diseased trees should be removed to eliminate source of infection and also for early salvage of heartwood, which becomes more and more decayed if trees are left in the stand.

Khair is indigenous in India, Pakistan, and Burma. It is tried elsewhere as an exotic. Importation of wood decayed by the fungus should be forbidden.

Hosts: Leguminosae—*Acacia catechu* Willd.

Literature: Bakshi, B. K. Diseases of khair (*Acacia catechu* Willd.) and their prevention. Indian Forester 83: 41–46. 1957.

Sal Heart Rot

Fomes caryophylli (Rac.) Bres. A heart rot fungus in high and coppiced sal forests. Infection occurs mostly through branch stubs, broken branches, and knots. Trees grow apparently healthy and may be identified by sporophores, or punk knots ("eyes"), which, however, are not constantly present. Wounds, swollen bole, and calloused knots may indicate heart rot, though not invariably so, and are thus rated on percentage basis. Decay in heartwood is white and spongy. Sporophores are small, dull brown, develop as thin crusts, not easily noticed.

Attacks young and mature trees. Up to 30 percent of the trees in the stand may be infected. Associates of sal and other hardwoods are also attacked.

The control is mainly based on prevention of injury which exposes heartwood to infection. Controlled burning, thinnings, and selection fellings with a view to stand improvement, adjustment of canopy to prevent frost, and suppression and formation of branch stubs are some of the measures suggested to reduce the incidence of the fungus. Infected coppice stands should be changed over to a crop of seed origin.

The pathogen occurs on many other hosts.

Hosts: Dipterocarpaceae—*Shorea robusta* Gaertn.

Literature: Bakshi, B. K. Heart rot in relation to management of sal. Indian Forester 83 : 651–661. 1957.

DANGEROUS FOREST DISEASES IN YUGOSLAVIA

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Leaf and Twig Blight of Poplars

Venturia populina (Vuill.) Fabr. (Imperfect stage: *Pollaccia elegans* Serv.). This disease attacks young leaves and shoots of different poplars, causing blackening and withering of these organs in the spring (usually May in southern Europe). The common Italian name for the disease is "Defogliazione primaverile" (Spring defoliation).

The fungus.—Brown hyphae on the infected organs bear brownish, mainly two-septate, or less frequently three- to four-septate, large conidia (28–38 × 10–11μ), roughly elliptical and mostly constricted at the septa. Perithecia, averaging 150μ in diameter, appear as the winter form of the fungus. They are sunken in the leaf tissue and have a neck with a hairy ostiole. Asci 110–190 × 19–23μ. Ascospores are light brownish, slightly swollen, 22–27 × 13μ, and commonly constricted at the septum which divides the spore into two uneven cells.

Symptoms.—The first symptoms are large, blackish, angular necrotic areas on young leaves and frequently on shoots. These necrotic areas are soon overgrown by olive-brown mycelia bearing conidia. This stage of attack is followed by a total withering and blackening of the above organs and a curving of affected succulent shoots in the form of a hook. The whole symptom picture resembles frost or fire killing. Complete defoliation occurs in severe attacks.

Age of hosts.—Young poplars in nurseries and plantations are especially susceptible. Areas with a high relative humidity, especially along rivers, and with frequent spring rains provide the best conditions for the development of this parasite.

Possibility of spread.—Intercontinental spread of the fungus may take place through shipment of unsterilized cuttings from infected localities or through infected leaves that are occasionally put into packing containers. Insects might also be fungus vectors.

Range: The disease is particularly common in Italy, but it also occurs in central Europe, Germany, France, and recently in Yugoslavia.

Hosts: The following are reported as the most susceptible poplars: *Populas nigra*, *P. nigra-italica*, and different cultivars of euramerican poplars. Italian clones proved to be highly resistant, although under favorable conditions for the fungus, clone I-214 was found to be intensely attacked (Yugoslavia). *P. alba* and *P. tremula* do not suffer from this organism. Reduced and stunted growth, top deformation, and bushlike appearance of trees are the most common results of the attack. Killing of young plants, though rare, is possible under heavy infections.

Literature:

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F.A.O. Poplars in forestry and land use. Rome, 1958: 326-327 (Section: Diseases of poplar, T. R. Peace, United Kingdom) 1958.

Servazzi, O. La defogliazione primaverile dei pioppi. Boll. Lab. R. Asservat. Fitopat. Torino. 1935.

Viennot-Bourgin, G. Les champignons parasites des plantes cultivees. T. II, Paris: 541–542. 1949.

Leaf Rust of Poplars

Melampsora allii-populina Kleb. This is a heteroecious rust occurring mainly on leaves of black and balsam poplars. Uredia appear, depending upon weather conditions, in mid-June and July on the under surface of poplar leaves. Uredospores are elongated to oval, one-celled, 23–38 × 11–18μ, thick-walled (2–4μ), spiny, yellow orange in color, catenulate. Paraphyses are thick-walled (2–3μ), capitate or rarely clavate, 50–60μ long, the swollen top measuring 14–22μ and the stalk 3–5μ in diameter. Telia in the form of dark-colored crusts appear on the undersurface of poplar leaves at the beginning of the winter season. Teliospores are one-celled, prismatic, rounded in their upper part, 35–60 × 6–10μ, with rather thick (1–2μ) and smooth walls. They germinate the following spring in a probasidium, 20–40 × 7–11μ, which later becomes four-celled, each cell producing a basidiospore. Pycnial and aecial stage are found on different *Allium* species in the early spring. Aecia are of the caeoma type. The aeciospores are one-celled, rather thick-walled (1.5–2μ), oval, 15–20 × 10–15μ, with gently roughened walls, orange red in color.

The following are symptoms of the disease: (1) Bursting of rust-colored pustules (uredia) on the underside of leaves on lower branches, from mid-June in southern Europe and in July farther north. New uredia appear during the growing season on the leaves of the upper portion of the crown. The rusty color is due to uredospores. (2) Building up of brownish crusts on fallen poplar leaves in autumn and winter, also on the underside. These crusts comprise the telial stage. (3) Orange-reddish color of leaves and stems of *Allium* spp. in the spring. This color is due to the formation of aecia with aeciospores. (4) Premature defoliation of poplars, starting usually at the end of August in the case of heavy attacks. Trees of practically all ages are susceptible. Areas with a high relative humidity are the most favorable for the spread of the fungus. Poor or inadequate soils seem to offer better conditions for the evolution of the rust.

There are three main disturbances of poplar plants resulting from the fungus invasion: first, a considerable reduction in growth both in diameter and height; second, frequently reported is a lowered resistance of diseased individuals towards other pathogens, such as *Dothichiza populea*; and finally, the defoliated shoots are more sensitive to autumn frosts and are therefore frequently killed. Dying back of poplars because of the rust is very rare and occurs primarily after consecutive heavy attacks on dry sites.

Possibility of spread.—From the intercontinental point of view, the spread of this rust is unlikely though it can take place through ship-

ment of poplar and *Allium* plants, as well as through flowers and seeds originating from infected plantations. Transference by unsterilized cuttings is also a possibility.

Range: This parasitic organism is present all over Europe but especially heavy infections of poplars have been recorded in the southern and central parts.

Hosts: Many species, cultivars, and clones belonging to the sections Aigeiros and Tacamahaca may be attacked. The appended table shows the relative susceptibility of many poplars but variations from this listing are possible. Susceptibility depends upon the origin of reproductive material as well as upon geographic location and climatic conditions. Among the widely used euramerican cultivars, cv. "serotina" proved to be one of the most susceptible.

POPLAR SUSCEPTIBILITY AND ALTERNATE HOSTS FOR MELAMPSORA ALLII-POPULINA KLEB.

Species aud resistance	*Alternate hosts*
Low:	
Populus nigra-italica	
P. thevestina	
P. berolinensis	
P. regenerata	
P. serotina	
P. backelieri	
P. monilifera	
P. robusta	
P. vernirubens	
Medium:	*Allium ascalonicum*
P. marilandica (Yugosl.)	*A. carenatum*
P. fremontii	*A. cepa*
P. balsamifera	*A. moly*
P. gelrica	*A. oleraceum*
P. wislizenii	*A. sativum*
P. nigra (Yugosl.)	*A. schoenoprasum*
P. deltoides	*A. spaerocephalum*
I–455 (Yugosl.)	*A. ursinum*
I–476 (Yugosl.)	*A. vineale*
High:	
P. nigra (Czech.)	
P. marilandica (Czech.)	
I–455 (Czech.)	
I–501	
I–159	
I–45/51	
I–214	

Literature:

F.A.O. Poplars in forestry and land use. Rome, 1958: 330–333 (Section: Diseases of poplar, T. R. Peace, United Kingdom).

Gremmen, J. Op Populus en Salix voorkomende *Melampsora-soorten* in Nederland. T. Pl. ziekten, 60: 243–250. 1954.

Leontovyc, R. Napadnutie jednotlivych klonov topolov hrdzou *Melampsora allii-populina* Kleb. v. selekcnej velkoskolske Gabci-

kova roku 1956. (Infections of different poplar clones by the rust fungus *Melampsora allii-populina* Kleb. in the selection nursery of Gabcikova in 1956). Les. cas., IV, 1 : 30–45. 1958.

Vujic, P. Problem Melampsora i drugih obolenja lista i borba protiv njih u plantazama topola (Problem of *Melampsora* and other leaf diseases including measures against them in poplar plantations). Mimeopr. Savet. centar polj. i sum., Beograd, 20 pp. 1959.

Bacterial Canker of Ash

Pseudomonas fraxini (Brown) Skoric (Syn.: *Bacterium savastanoi* Smith var. *fraxini* Brown). This pathogen causes numerous perennial stem and branch cankers of ash in Europe. Cankers are mostly of a sunken and less frequently of a knobby type. Very young cankers appear as one or two splits in the periderm and are followed by cross-splits and openings of the bark. In the more advanced stage the bark tissues become definitely broken down to the sapwood, imparting a blackish or brownish color to it. At the same time, the bark bordering the canker begins to grow more vigorously, resulting in the formation of swellings and the enlargement of callous rings around cankers. Small cankers of the sunken type are formed in 1 to 2 years and large ones, reaching 10 to 20 cm. in diameter, in 10 to 30 years.

Trees of all ages are susceptible. The disease is common in pure or mixed ash stands, in parks, and along avenues. Valleys and localities with a high relative humidity offer the best conditions for the development of cankers. It is believed that the causal agent spreads mainly by rain and probably by the common ash beetle (*Leperisinus fraxini* L.) or even by mites. Wounds of any kind are the main entries through which bacteria penetrate into cortical tissues.

Deformations of the stem, stunted growth, and bushlike appearance of attacked individuals are the principal kinds of damage. Esthetic value of affected trees grown as ornamentals is reduced. Mortality of young plants may result from rapid stem girdling.

Possibility of spread.—Intercontinental spread is possible through shipment of infected young plants and unbarked logs. The organism may spread on unsterilized ash seeds originating in infected areas.

Range: The disease has been reported chiefly in Austria, France, Great Britain, Italy, Netherlands, and Yugoslavia.

Hosts: English ash (*Fraxinus excelsior* L.) is reported as the most susceptible species. White ash (*Fraxinus americana* L.) and green ash (*Fraxinus pennsylvanica lanceolata* (Borkh.) Sarg.) are also found to be attacked.

Literature:

Brown, N. A. Canker of ash trees produced by a variety of the olive-tubercle organism, Bacterium savastanoi. Jour. Agr. Res. 44:701. 1932.

Skoric, V. Jasenov rak i njegov uzrocnik (The ash-canker disease and its causal organism). Glasnik za sumske pokuse, Zagreb, 6: 66–97. 1938.

DANGEROUS FOREST DISEASES IN AUSTRIA

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Introduction

The economic and international importance of forest diseases must be judged from two different points of view.

On the one hand, there are diseases which attack individual trees and cause stationary damage. This is accepted as a fact. Should these diseases be widespread in neighboring countries too, it need not be supposed that the significance of the diseases has suddenly increased. If, however, neighboring countries are completely free of these diseases and more favorable conditions for the diseases arise, then the situation is quite different.

On the other hand, there are forest diseases in every country which are well known but largely disregarded for two reasons—either (a) their economic importance is too slight or (b) the aggressiveness of the fungi is of no importance. Diseases of this kind, however, may become very dangerous if a sudden aggressive mutation takes place in the fungus.

In order to demonstrate conditions in Austria, it is necessary first of all to give a survey of the distribution of individual trees in this country.

	Hectare	*Percent*
Conifers:		
spruce	1, 824, 000	58. 1
white pine	305, 000	9. 7
fir	220, 000	7. 0
larch	211, 000	6. 7
Pinus montana	41, 000	1. 3
P. nigra	31, 000	1. 0
P. cembra	16, 000	0. 5
		84. 3
Broad-leaved trees:		
beech	300, 000	9. 6
oak	65, 000	2. 1
alder	31, 000	1. 0
hornbeam	31, 000	1. 0
poplar and willow	29, 000	0. 9
ash	12, 000	0. 4
maple	11, 000	0. 3
other broad-leaved trees	11, 000	0. 4
		15. 7

Among the great number of forest diseases which wield a permanent influence on Austria's economy, the following should be especially mentioned:

Armillaria mellea Vahl. ex Fr.
Chrysomyxa rhododendri De By.
Fomes annosus (Fr.) Cke.
Lophodermium pinastri (Schrad. ex Fr.) Chev.
Trichoscyphella willkommii (Hart.) Nannf.
Damping-off, which causes damage in forest nurseries.

It is worth noting the unimportance of chestnut blight (*Endothia parasitica* (Murr.) P. J. & H. W. And.) in Austria.

In the author's opinion the following diseases are of international importance and should be discussed in detail:

Cenangium ferruginosum Fr.
Chondroplea populea (Sacc.) Kleb.
Guignardia aesculi (Peck) Stewart
Phacidium infestans Karst.

Dieback of Pines

Cenangium ferruginosum Fr. (German term: Kieferntriebsterben.) In the spring and summer of the year 1960 *Cenangium ferruginosum* appeared particularly among *Pinus nigra* in Austria. This fungus is usually a saprophyte. It occurs occasionally as a parasite and then wreaks heavy damage. In 1956, Spaulding explained the cause of this disease as lack of water or dryness. The outbreak of the disease is also furthered by insect injuries. Thus the trees must already be weakened.

The following dates can be found in literature for the epidemic occurrence of this fungus on *Pinus sylvestris* and *P. nigra:*

1891/93 in North Germany
1926/28 in East Prussia
1933/34 in North and Central Germany
1928/29, 1939, 1948, and 1960 in Austria

The last devastating occurrence of this fungus in Austria was during the spring and summer of 1960. It affected all the regions containing *Pinus nigra.* The first symptom was the discoloration of the needles near the tree top. The color of the needles was yellowish gray to brownish ochre and the discoloration started from the base of the needles. In many cases the terminal buds dried up and in a comparatively short time the tree died. The infection must have started in autumn or early spring. The mycelium infects the bark and causes the destruction of the youngest shoots. The diseased trees can easily be detected at a great distance. Pine trees from the fifth year onwards are liable to infection by this fungus.

The disc-shaped fruit bodies—apothecia—usually burst out in quantities near the needle-bed and their color varies from yellowish brown to black. The apothecia have a diameter of 1.5 to 3 mm., the club-shaped asci are rounded off at the top (60 to 80μ long, 10 to 12μ wide, eight spores in an ascus). The length of the spores is from 10 to 12μ, their width from 5 to 7μ and the paraphyses are thickened at the top.

Austrian foresters mistakenly call this fungus "*Brunchorstia pini*," according to a paper by Donaubauer (1960). All reports on "*Brunchorstia pini*" in Austria actually refer to *Cenangium ferruginosum*.

This disease is very dangerous for *Pinus nigra* and *P. sylvestris* and it is expected that it will also spread to other pine trees. The damage it caused in Austria was 15,000 fm. (or 4.5 million Austrian schillings) in 1948.

Control measures.—To prevent this disease from spreading, the diseased trees are felled and the infected branches burned.

Range: Europe, America.

Hosts: Pine trees—

Pinus attenuata
P. contorta
P. nigra
P. rigida
P. sylvestris
P. strobus

Literature:

Donaubauer, E. Die Kieferntriebsterben-Kalamitat 1959/60. Allgemeine Forstzeitung, 71, Jg., Folge 9/10. 1960. (Informationsdienst, 32. Folge).

Spaulding, P. Diseases of North American forest trees planted abroad. Agr. Handb. 100, U.S. Dept. Agr. 1956.

Poplar Canker

Chondroplea populea (Sacc.) Kleb. (German term: Pappelrindentod, Pappelrindenbrand.) This serious disease of poplar has caused heavy damage in Austria—especially in Lower Austria—during the last few years and is, unfortunately, always erroneously termed "*Dothichiza populea* Sacc. & Briard" in phytopathological literature on forestry.

The disease is found in the crown of old trees and from there it attacks more recent plantations. Very much endangered are afforestations overgrown with weeds (*Solidago canadensis*). The general opinion now is that the infection attacks the pits or wounds in the bark. It kills the bark and the cambium. Macroscopically such places are detected by distinct indentations in the bark. According to the size of the part infected and the amount of cambial area destroyed, the plant dies off sooner or later.

Butin (1957) holds the view that the further away the cortical cells are from water saturation, the more quickly the fungus spreads in the cortical tissue. In addition he considers that mild winters further this disease, since the fungus can go on growing while the plant is unable to produce any defensive reactions during hibernation.

In spring and summer, pycnidia break out through the infected bark and empty the spores in the shape of cirri. The diameter of the pycnidia varies from between 500μ to 1000μ. The hyaline spores are 10 to 13μ long, 7 to 9μ broad, and stand on a single base.

Control Measures.—According to research carried out in Austria it has been found that when afforesting with poplars it is advisable to have a rotation of, for instance, maize, turnips, cabbage, or potatoes in between. This cultivates the soil and checks the growth of weeds. At the same time it reduces the humidity of the air and checks the germination of spores. Control measures by means of recently

developed fungicides are only possible in forest nurseries. A tree which successfully resists *Chondroplea populae* has not yet been cultivated.

Range: Europe, Canada, North America, Argentina.

Hosts: On the susceptibility to this disease of various kinds of poplars, Butin (1957) states the following:

> The available data in existing literature on various kinds of poplars susceptible to this disease stress an increased susceptibility of those trees belonging to the Aigeiros group. Of this group *Populus* cv. *robusta*, *P.* cv. *bachelieri*, *P.* cv. *eugenei*, *P. nigra*, var. *italica*, and *P.* cv. *vernirubens* are obviously in grave danger, and *P.* cv. *marilandica* and *P. balsamifera*-group, but to a much lesser degree. *P. alba*, *P. tremula*, *P. tremuloides*, and *P. candicans* are mentioned as resistant or less endangered species.

Literature:

Butin, H. Untersuchungen uber resistenz and Krankheitsanfolligkeit der Pappel gegenuber Dothichiza populea Sacc. et Br., Phyopath. Ztschr. 28:353–374. 1957.

——— Die blatt—und rindenbewohnenden Pilze der pappel unter besonderer Berucksichtigung der Krankheitserreger. Mitt. aus d. Biol. Bundestanst. f. Land–u. Forstw. Berlin-Dahlem, Heft 91: 50–52. 1957.

Petrak, F. Uber die Gattungen Dothichiza Lib. und Chondroplea Kleb. Sydowia, Ann. Mycol., Vol. X: 201–235. 1956.

Leaf Blotch of Horse Chestnut

Guignardia aesculi (Peck) Stewart (German term: Blattrollkrankheit der Rosskastanie, Blattbraune der Rosskastanie). This disease was minutely described by Stewart (1916). According to his report it causes great damage to *Aesculus hippocastanum* and *A. glabra* in America.

In recent years this disease has spread more and more in Europe and caused considerable damage. The latest paper on leaf blotch of horse chestnut was written by Petrak in 1956.

The macroscopic symptoms may be summarized as follows: Reddish-brown blotches of considerable size appear and spread quickly over the leaves, which then die. At the same time the leaves roll up cornet-wise, which accounts for the German word for this disease, namely: Blattrollkrankheit.

Petrak also points out the fact that there are still some healthy trees even in badly infected areas. By the propagation of these trees the disease could be checked.

Through microscopical examination of infected leaves, Petrak discovered that loosely spread pycnidia of *Phyllostictina sphaeropsoidea* (Ell. & Ev.) Petr. were contained in the blotches on the leaves that were still green. In the dead leaves the examination revealed pycnidia of *Asteromella aesculicola* (Sacc.) Petr. and the related perithecia of *Guignardia aesculi* (Peck) Stewart.

Phyllostictina sphaeropsoidea. Pycnidia: Roundish or broad-elliptical 80 to 170μ, occasionally up to approximately 200μ in diameter. Conidiospores 10 to 15μ long and 6 to 10μ broad.

Asteromella aesculicola. Pycnidia: Mostly 40 to 80μ, conidiospores 4–6 × 1–1.5μ big; in some pycnidia even bigger: 5 to 9μ long and 1.5 to 2.5μ broad.

Guignardia aesculi. Perithecia: 80 to 170μ, Asci 54–70 × 15–17μ big, ascopores elliptoidic 12–18 × 7–9μ big.

Control measures.—Collecting and burning the infected leaves in autumn. Breeding of resistant species.

Range: Europe, America.

Hosts: *Aesculus hippocastanum*—
A. glabra
A. pavia

Literature:

Petrak, F. Uber ein verheerendes Auftreten der Blattrollkrankheit der Rosskastanie in der sudlichen Steiermark. Sydowia, Ann. Mycol. Vol. X (1–6) : 264–270. 1956.

Stewart, V. B. The leaf blotch disease of horse chestnut. Phytopathology VI : 5–19. 1916.

Snow Blight of Pines

Phacidium infestans Karst. (German term: Weisser Schneeschimmel an Nadelhölzern.) In Austria every year heavy damage is done by avalanches, including the death of many people. For this reason an effort is made to afforest the steep slopes with *Pinus cembra* L. Whereas the snow melts quickly on the southern slopes, it takes a very long time for the northern slopes to become free from snow. The slow melting of the snow creates good living conditions for the fungus *Phacidium infestans* which infects the young *Pinus cembra.* According to the size of the tree it impedes growth and development and may even cause death. As Björkman ascertained (1942 and 1948), this fungus is able to grow actively under a covering of snow at a temperature of minus 5° Celsius. The significance of this fungus as the pathogenic agent for *P. cembra* in Austria was discovered in 1955.

The fungus was first described in 1886 by Karsten, who found it in Finland. Until now this fungus has been found only on pine trees in the northern parts of Europe, Asia, and America. The infection of the trees takes place through the needle stomata in autumn. The first pathological symptoms can be seen in spring as soon as the thaw has started. The needles become brownish. Under the influence of the sunlight they take on a paler color in summer and become greyish toward autumn. The needle cast does not take place the first year after infection. The apothecia do not develop before July, by which time small black dots can be seen on the needles. The apothecia are not fully grown till October.

Control measures.—No practical controls known.

Range: Europe, America, Asia.

Hosts: Pines

Literature:

Björkman, E. Renkulturforsök med snöskyttesvampen (*Phacidium infestans* Karst.) Sv. Bot. Tidskr. 36. 1942.

———. Studie over snöskyttesvampens (*Phacidium infestans* Karst.) biologi samt metoder for snöskyttets bekämpande. Medd. f. Stat. Skogsforkningsinst. 37, 2.

Petrak, F. Uber *Phacidium infestans* Karst., einen gefahrlichen Parasit der Zirbelkiefer und einige andere in seiner Gesellschaft wachsende Pilze. Sydowia, Ann. Mycol. IX: 518–526. 1955.

COMMENTS ON SNOW BLIGHT

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The *Phacidium*-blight fungus, belonging to the family Phacidiacae of the Ascomycetae, was first described in Finland by Karsten in 1886 under the name of *Phacidium infestans* Karst. The fungus is known from the northern parts of Europe and Asia down to southern Ural. It has also been reported from North America (Faull 1929). In Europe and Asia this fungus appears almost exclusively on *Pinus* species, especially *Pinus sylvestris*, whereas in the northern parts of the U.S. and in Canada, it appears commonly on a great number of conifers, especially *Abies* and *Picea* species.

From what has been found by the infection experiments hitherto undertaken, it appears that the type called *P. infestans* Karst. in America is not, at least physiologically, quite identical with the European and Asian type. As it seems chiefly to attack *Abies* species, it is proposed that this fungus for the time being be called *P. infestans* var. *abietis*, as it was once called by Weir.

The ascospores of *P. infestans* are hyaline, kidney-shaped, 16 to 23μ long and 6 to 8μ wide.

The spores of the fungus disseminate in the autumn and germinate as soon as the needles become embedded in snow so that the fungus receives the required moisture. Snow blight attacks needles in all ages, consequently also living needles on old windfalls on the ground. Generally the dissemination takes places within a restricted area which, however, can be increased considerably under favorable conditions. During germination a grayish-white mycelium spreads on the needles.

The living conditions of the snow-blight fungus: Among the conditions of life decisive for the development of the snow-blight fungus, temperature is the most important one. A series of growth experiments showed that the optimum temperature for the fungus' development was +15° C. At a temperature of +20° C. the mycelium grows at a considerably much lower rate than at +10° C., and at a constant temperature of +25° C. it dies after a certain length of time. Growth is still good at +5° C. and even at 0° C. Growth has been demonstrated at a temperature as low as −5° C. (Björkman 1948).

The depth of snow cover and the temperature in it. A snow cover of great compactness—which occurs after the action of thaw—conducts the cold better than a snow cover that has never been affected by temperatures above zero. In small openings and small cutting areas the depth of the snow cover is generally greater than in large clearings. Close to the trunks of old seed trees the depth of snow is always less than between the trees.

The snow-blight fungus may, with regard to temperature conditions only, grow in snow up to a height of 1 to 2 dm., even during the coldest period of midwinter.

The resistance of different pine provenances against snow-blight attacks. Experiments, conducted in a special "snow-blight nursery," consisting of fastening snow-blight mycelium to pine seedlings of various origins, show that plants of a northern provenance are more resistant to snow blight than those of a southern provenance—a result agreeing with old observations of spontaneous attacks in pine cultures. According to Langlet (1936), there is an apparent connection in the way that needles on plants of northern origin with a higher dry substance (and sugar content) are not attacked to the same degree as needles on plants of a more southern origin with lower dry substance.

Literature:

Björkman, E. Studies on the biology of the *Phacidium*-blight (*Phacidium infestans* Karst.) and its prevention. Medd. Stat. Skogsforskn. inst. 37. 1948.

Faull, J. H. A fungus disease of conifers related to the snow cover. Jour. Arnold Arboretum 10. 1929.

Langlet, O. Studien uber die physiologische Variabilitat der Kiefer und deren Zusammenhang mit dem Klima. Beitrage zur Kenntnis der Okotypen von *Pinus silvestris* L. Medd. Stat. Skogsfors. anst. 29. 1936.

DANGEROUS FOREST DISEASES IN WESTERN EUROPE

Scots Pine Blister Rust

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Cronartium flaccidum (Alb. & Schw.) Wint. is very similar in morphology, biology, and disease symptoms to *C. ribicola*, the cause of white pine blister rust. It forms perennial cankers on the stem and branches of pines. Resin flow and dark color of the bark are characteristic. Where the bark is killed, the growth stops, and the canker becomes more or less flattened. But above and to the sides of the canker the growth is usually greater than normal. Small branches and twigs may get spindle-shaped swellings. The cankers grow from year to year, and much faster in the longitudinal direction than around the circumference. Small stems may become girdled in a few years, big stems after 10 to 50 or more years.

Spermogonia are flattened, inconspicuous, 2 to 3 mm. in diameter. Aecia are formed from May to July, along the attacked bark of twigs, and at the border of older cankers. They are blisterlike, 2 to 8 mm. long, 2 to 3 mm. wide, and 2 to 4 mm. high. The wall is thin and hyaline, the yellow-orange color of the spores showing through the wall. Peridium cells are 15 to 40μ in diameter, the walls are verrucose and 4 to 6μ thick. Aeciospores are roundish—ellipsoidal, or somewhat polyhedral, 22–31 × 15–22μ. The greater part of the wall is 3–4.5μ thick and coarsely verrucose; the rest of the wall is thinner, 2–3μ thick, nearly smooth, because the warts are much lower. The wall itself is colorless, but the content gives the spores a yellow-orange color. Uredo- and teliospores are formed on the dicotyledonous hosts on the lower side of the leaves. Uredia are small, up to 0.25 mm. in diameter, pustular. The peridium has a small opening at the top. The uredospores are ellipsoidal or obovoidal, 20–27 × 15–20μ; the wall is spiny; the pores are inconspicuous. Telia are columnar—filiform, 0.4 to 2 mm. long and 0.04 to 0.13 mm. in diameter, horny when dry; they are formed in older uredia. Teleutospores are elongated ellipsoidal, the ends more or less truncated, 25–56 × 9–14μ. Basidiospores are roundish, about 8μ in diameter.

Life history.—Scots pine blister rust embraces one host-alternating and one nonalternating race. The host-alternating race (heteroecious) attacks as haplont some 2-needled pines. The dicaryophyt is very pleophag, as shown in the host list. With *Gentiana asclepiadea* as differential host, the host-alternating race can be divided into two formae speciales: F. sp. *typica* attacks the greater part of the dicotyledonous genera, but not *Gentiana*. F. sp. *gentianea* attacks *Gentiana*, but not *Cynanchum*. *Paeonia* is attacked by both the mentioned formae speciales.

The nonalternating race (*Peridermium pini*) has repeating aecia. There may be some very small morphological differences between the aecia of the host-alternating and those of the nonalternating race. But even if there are slight differences, they are too small to serve as distinctions between the two races. In Norway and Sweden, at any rate, the nonalternating race is by far the most common.

Resistance.—As to resistance, there is a considerable difference between individuals of Scots pine. The resistance has been proved to be inheritable.

Importance.—In Europe and Northern Asia *Cronartium flaccidum* is one of the most important pathogens on two-needled pines, especially Scots pine. Stands with a high percentage of killed or spike-topped trees, as a result of attacks by this blister rust fungus, are not infrequent.

Control.—Control measures are—

1. Eradication of the alternate hosts
2. Removal of infected trees
3. Use of seed from resistant trees

Potential threat.—We do not know which of the two-needled pines may prove susceptible, but one or more of important American species may be damaged if the host-alternating or the nonalternating race should be spread to America.

Range: Nearly all of Europe. Northern Asia to the Far East, Korea, and Japan.

Hosts: Pinaceae (section Diploxylon). Aecial hosts—

Pinus densiflora Sieb. & Zucc.
P. halepensis Mill.
P. mugo Turra.
P. nigra Arn.
P. pinaster Sol
P. sylvestris L. (most important host)

Uredo and teleuto on many dicotyledonous genera in widely different families—

Acanthaceae: *Ruellia*
Asclepiadaceae: *Asclepias*, *Cynanchum*
Balsaminaceae: *Impatiens*
Gentianaceae: *Gentiana*
Loasaceae: *Grammatocarpus*, *Loasa*
Ranunculaceae: *Paeonia*
Solanaceae: *Schizanthus*
Scrophulariaceae: *Euphrasia*, *Melampyrum*, *Nemesia*, *Pedicularis*
Tropaeolaceae: *Tropaeolum*
Verbenaceae: *Verbena*

Literature:

Gäumann, E. Die Rostpilze Mitteleuropas: 81–84. Bern. 1959.
Kuprevicz, V. T., and Tranzschel, V. H. Flora plantarum cryptogamarum USSR Vol. IV. Fungi (1). Uredinales. Fasc. 1. Familia Melampsoraceae: 261–265. Moscow, Leningrad. 1957.

Larch Canker and Dieback

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Dasyscypha willkommii (Hart.) Rehm. A perennial—and often girdling—stem and branch canker of larch in Europe.

First symptoms are regular, elliptical to nearly circular bark depressions, in the majority of cases a dead dwarf shoot or, sometimes, twig in the center. Resin flow is often pronounced. Older, crusty bark is not attacked unless through a branch already cankered. In summer a callus formation takes place, the necrotic bark becoming closed off from healthy tissue by a cork layer. During the resting season of the tree, the fungus passes this layer and a perennial canker results. After several years, large almost amphitheatrical, wounds develop on the stems and coarser branches; while at the same time a more or less conspicuous convexity of healthy wood is being formed on the opposite side of the axis. A younger axis is easily girdled; smaller trees are thus killed in almost their whole length, somewhat larger trees getting lots of died-back branches and twigs, and young- to medium-aged stands often declining as a result of the attack.

In tiny, yellow-white pustules on young cankers are produced minute, hyaline, unicellular conidia—their role obscure, however, because germination is never observed. Later they develop 1 to 4 mm. broad, cupshaped or saucershaped apothecia, which are whitish with orange-red hymenium. Asci are cylindrical, octosporous, with spores hyaline, oblong to elliptical, normally unicellular, their extremities often somewhat pointed, size ca. 14–30 × 6–10μ.

Most probably ascospores infect mainly through recently dead or weakened dwarf-shoots or twigs. Other ways of entrance may exist, among them, perhaps, suction canals of aphids.

The part played by the fungus in the development of the disease is not always evident; however, the following facts deserve attention:

1. *Dieback* can occur without any cankers being observed.
2. There are indications that spontaneous cankers of the said type can develop also in absence of the *fungus*.
3. In spontaneously developed cankers the callus shows cell structures commonly identified as frost rings, and numerous observations confirm the importance of frost to initiation and further progress of the canker formation.
4. Cankers have been produced by artificial freezing, no *Dasyscypha* being present.
5. Cankers have been produced also by inoculation of *Dasyscypha* mycelium into artificial wounds under conditions free of frost.
6. In most countries canker and fungus are almost generally associated.
7. Several investigators, among them the subscriber, agree that purely saprophytically growing *Dasyscypha* strains are distinct from those growing in cankers.

The main host of larch canker is *Larix decidua* Mill. All geographical varieties are included, spontaneous forests showing sporadical and

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moderate attack only. Larch cultivated outside its native range of distribution often is very heavily attacked, susceptibility increasing, broadly speaking, with sinking continentality of the culture spot and varying, to a considerable degree, with provenance.

These facts all considered, the following conclusion seems justified. *Dasyscypha willkommii* is a pathogen on *Larix decidua*. Though of minor importance within the native range of the tree, it becomes more important the more the culture conditions differ from those offered by the home countries, unless climate and site are so unsuitable to the tree that the cultures decline as a consequence of these adverse conditions alone.

Hosts (in Europe): Pinaceae	*Susceptibility*	
Larix decidua Mill	Great	
L. sibirica Ledeb. (including *L. suchaczewii* Dyl.)	Great	
L. leptolepis Gord	None or low	
L. gmelini Pilger sens. lat	Moderate to medium	Observation material relatively restricted
L. olgensis Henry	Moderate to medium	
L. laricina Koch	Great	
L. occidentalis Nutt	Great	
L. decidua × *L. leptolepis:* F_1 as a rule greatly resistant, F_2 variable.		

Intercontinental spread possible mainly through shipment of infected trees or cuttings.

Chances are great that, introduced into other areas of the world, the disease will be important mainly to larches cultivated outside their native growth range, though the possibility cannot be excluded that some larch species possesses a general susceptibility, just as *L. leptolepis* is almost generally immune or very resistant.

Literature:

Hartig, R. Die Lärchen-krankheiten, insbesondere der Larchenkrebspilz, *Peziza willkommii* m. Untersuchungen aus dem forstbotanischen. Inst. Muchen: 63–87. 1880.

Forestry Comn. Leaflet 16, London, Revised 1948.

Hypodermella Needle Cast of Pine

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Hypodermella sulcigena (Rostr.) Tub. A needle cast disease occurring on two-needle pines in Europe.

First symptoms occur in late summer and early autumn, the current year's needles turning smoke grey to pale reddish violet, their basal end often remaining green, sharply distinguished from the discolored tissue. Only a few needles or all needles per annual shoot are attacked. During autumn and winter, needle color changes to pale yellow brown, interior resin exudations causing formation of dark dots or irregular specks. Needles killed from the base are dropped. Withered needles do not stiffen.

In attacked needles remaining on the shoots are formed apothecia which in spring become easily visible in the shape of 2 to 15 mm. long, narrow, grey-brown to grey-black ribs on the lower side of the needles. The numerous cylindrical asci contain 8 (rarely 4) hyaline, unicellular spores, elongated droplike or obliquely club-shaped, each measuring 30–50 × 4–6μ and being surrounded by a slime mantle. Apothecia open in May or June by a longitudinal split. Discharged spores infect new needles. Killed old needles, then pale reddish grey to almost white in color, are dropped during summer.

The infection period in spring is short—infection apparently successful only in juvenile needles after abundant rain. Attack is often very uneven within the stand, partly owing to variations in time of flushing. Marginal trees and trees in openings are most frequently attacked. Though all age classes are susceptible, 10- to 30-year-old individuals are as a rule most easily infected.

Both plantations and spontaneous forests are attacked.

Because only the current year's needles are infected, trees are never reported killed. Repeated attack several years in succession causes stunted growth with abnormally short shoots.

The disease is reported mainly from the temperature zone of Scandinavia, being mentioned also from Scotland and the Pyrenees (and other parts of Spain). In Germany it is probably frequent although there it is most often attributed to attack by a pycnidiferous fungus *Hendersonia acicola* v. Tub. with brown, elongated, ellipsoidical, 3-septated spores. This fungus is often, incorrectly, considered the conidial stage of *Hypodermella sulcigena*. In Scandinavia *Hendersonia* occurs as a secondary fungus in needles attacked by *Hypodermella*, and its presence seems to depress the apothecial formation of the last-named.

Hosts: Pinaceae—

Pinus sylvestris L.
P. mugo Turra
P. nigra var. *austriaca* Asch. & Graebn.
P. halepensis Mill.
(A disease on this species in Spain recently mentioned to me by Dr. J. B. Martinez, seems to be identical with ours.)

Intercontinental spread possible through shipment of plants, trees, and cuttings.

Literature:

Lagerberg, T. Om gråbarrsjukan hos tallen, dess orsak och verkningar. "Die Hypodermella-krankheit der kiefer und ihre Bedeutung." Meddel. f. Statens Skogsførsøksanst. 7-8, Stockholm 1910.

Rostrup, E. Fortsatte undersgelser over Snyltesvampes Angreb paa Skovtrserne. Tidskr. f. skovbrug, V. 6, Copenhagen 1883.

Pine Twist Rust

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Melampsora pinitorqua Rostr. is a heteroecious fungus developing its pycnial (spermogonial) and caeomatal (aecial) stage on *Pinus*

species and uredinial and telial forms on poplar. A detailed account of host range will be discussed in this short contribution.

In contrast to other *Melampsora* species like *M. larici-populina* Kleb. and *M. larici-tremulae* Kleb. causing premature defoliation of poplar leaves, *M. pinitorqua* brings about considerable damage to leaders of pines which become distorted and even may be killed. Shoots of the current season are infected by basidiospores (sporidia), giving existence to colorless pycnia (0), afterwards succeeded by orange-yellow caeomata (I). In summer uredinia (II) and telia (III) are produced on the alternate host. After hibernation of the dead, fallen leaves these telia may germinate forming basidiospores.

In the period 1953–54, *Melampsora pinitorqua* affected stands of pine trees 5 to 7 years old grown in the nurseries of the Forest Research Station at Wageningen. In close proximity of these pine trees, poplars were cultivated, many of which consisted of aspen and crossings of aspen. From about the end of May the minute, colorless pycnia, 4 to 5 days afterwards followed by caeomata, were developed on the pine shoots. The number of caeomata considerably increased during the months of June and July.

Scots pine, mountain pine, and maritime pine appeared to be very susceptible to the rust, whereas Corsican pine and Austrian pine, as well as *Pinus rigida* Mill., demonstrated a striking resistance against the parasite. In these species very few infections have been observed. The following poplar species were grown in the vicinity: *P. tremula* L., *P.* × *canescens* Sm., *P. alba* L., *P. grandidentata* Michx., *P. tremuloides* Michx., and many other species of botanical interest of sections Leuce Duby, Aigeiros Duby, and Tacamahaca Spach.

In order to obtain more data on host range in the genus *Populus* L., a number of species and clones of Leuce poplar have been investigated. Caeomaspores of *Melampsora pinitorqua* collected from maritime pine have been used for inoculating discs of leaves floating on water in petri dishes. A second series of discs has been used for comparison and inoculated with caeomaspores of *Melampsora larici-tremulae*.

Host plant:	*Melampsora pinitorqua*	*Melampsora larici-tremulae*
Populus tremula, no. 9	++	+++
P. tremula, no. 23	++	+++
P. canescens, no. 34	++	−
P. alba, no. 31	+	−
P. alba, no. 37	−	+
P. grandidentata, no. 85	−	−
P. grandidentata, no. 88	−	+
P. grandidentata, no. 99	−	−
P. tremuloides	−	?
P. tremuloides, no. 78	++	−
P. tremuloides, no. 79	−	−
P. tremuloides, no. 96	++	++

Explanation: +++=very many uredinia; ++=many; +=only few; −=none.

Uredinial infection of the discs evidently demonstrated that *Populus tremula*, *P. canescens*, *P. tremuloides*, and less *P. alba* are hosts

of the rust; whereas *P. grandidentata* is out of host range at least for this rustrace.

Some years afterwards the number of infections caused by the parasite decreased and finally ceased, although the situation remained the same in some parts of the nursery, except that pines as well as poplars became older, reaching the age of about 13 to 15 years. Consequently there is circumstantial evidence that infection chance in young stands is distinct from those in older stands.

In a related species, *Melampsora larici-populina*, we are often struck by the fact that needle infection of the larch is extremely abundant on branches hanging closely to the ground near old leaves carrying telia of the rust. The higher side branches show diminishing numbers of infections and after a certain distance from the ground the needles remain free from them.

This implies a limited spread of these very minute, hyaline, thin-walled basidiospores, which is in contrast to the uredospores which are thick-walled, spiny or warty, and suitable for dispersal by men, animals, and wind over long distances.

In case of *Melampsora pinitorqua*, explanation as to the disappearance of infections on pine seems to be basidiospore dispersal.

Spread of the leaves carrying the telia over long distances is only possible by wind. After establishment of this inoculum the telia may germinate in next spring and infection of the alternate host is only then realized when:

1. A susceptible host is available.
2. A close proximity of this host is available (probably infection is only possible within a few meters).
3. When the host has a certain physiological condition (development of the shoot) making infection possible.

Range: Distribution throughout all countries of Europe and the western part of America.

Hosts:

Pinaceae—

Very susceptible:

Pinus sylvestris L.
P. montana Mill.
P. pinaster Sol.
P. ponderosa Laws. (in Canada)

Resistant:

Pinus laricio Poir.
P. nigra Arn.
P. rigida Mill.

Salicaceae—

Populus alba L.
P. canescens Sm.
P. tremula L.
P. tremuloides Michx.

Literature:

Moriondo, F. Richerche sulla Melampsora pinitorqua in Italia. Accad. Ital. Sc. Forest. 1956.

Murray, J. S. Rusts of British forest trees. Leaflet Forestry Comn., London, 4. 1955.

Sylvén, N. Om tallens Knäckesjuka. Medd. Stat. Skogsforsöksanstalt. 1917.

Top Canker of Spruce and Pine

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Scleroderris lagerbergii (Lagerb.) Gremmen. A parasite on *Pinus* in western Europe and on *Picea* mainly in Scandinavia. The identity of this fungus was elucidated by Lagerberg in 1913 when, among other things, earlier confusions with *Cenangium ferruginosum* Fr. were explained, and the pycnidial stage of the fungus, which had been described as *Brunchorstia pinea* (Karst.) v. Hohn, was classified under the specific name *Crumenula abietina*. This species has later been described by Gremmen and van Vloten in 1953 as *Scleroderris abietina* (Lagerb.) Gremmen, and by Gremmen in 1955 as *Scleroderris lagerbergii* (Lagerb.) Gremmen. Some other synonyms are: *Crumenula pinea* (Karst.) Ferd. & Jorg., and for the pycnidial stage *Brunchorstia destruens* Eriksson.

The fungus generally attacks young spruce, but under epidemic conditions may sometimes appear also on older individuals. The damage is generally discovered when the tops of the spruce die and, on a more close investigation, a wound or a clear zone between the living and the dead tissue on the top shoot of the year before is found. The infection will therefore probably take place on the top shoot itself, but it is first in the following year that the supply of nutrition is cut off causing the top of the spruce to die. A heavy resin flow appears near the zone between living and dead tissue where also the reproduction stages will be found.

The pycnidia are black, hemispherical, and 0.2 to 1.5 mm. in diameter. The apothecia are easily confused with the pycnidia, being about 1 mm. in diameter, but are as a rule recognized by their flat or concave tops. Through burstings in the pycnidium wall, the spores are pressed out, especially in wet weather. The spores are sickle-shaped with 2 to 6 cells (generally 4) and of varying size. Lagerberg estimates spore size to vary between $4 \times 43\mu$ and $3 \times 22\mu$. The spores germinate easily in water. The asci are elongate without an apparent stem, $100 \times 160\mu$ and $9 \times 15\mu$. The ascospores are fusiform with 4 cells. In typical cases the size will be 15–24μ and 4–6μ. The spores also germinate easily in water, even if still enclosed in the asci.

Spruce tops may be much deformed by the fungus; after a couple of years, however, the results of the damage will as a rule disappear. The fungus can be considered as being of relatively little importance as far as the spruce is concerned.

On *Pinus* the species has been described as "bud-drought," as it is the buds on the fully grown shoots that are attacked and then do not shoot in the following spring. This stage can develop into a "branch-drought." Especially on pines imported to Sweden it can have a quite devastating effect. The fungus has broadly been described in this way by Lagerberg, but it has never been necessary to reckon with very heavy damages on *Pinus sylvestris*.

In 1958, however, this fungus also started attacking 1-year-old and especially 2-year-old seedlings in a great number of nurseries in northern Sweden, and millions of seedlings were killed. Shoots and buds are infected during all summer and autumn, first through conidia and

later through ascospores. The attack is, however, not visible to the eye until the spring, when the snow on the plants melts away and they are exposed to the direct influence of the light. The damage is first noticed as brown spots on the shoot inside the needle base. The needles will then get characteristically brown from inside out towards their points, and finally the needles as well as the buds and the whole shoot will die.

The fungus seems to be a weak parasite that needs specially favorable conditions to spread epidemically. The reason for its occurrence in such a large scale in 1958 and the years after, not only in nurseries but also on pine heaths in upper Norrland, was probably the heavy thaw in February in 1958, by which the plants were directly damaged in the compact layer of ice that was left when the cold set in again in March the same year. Another important factor was that the plants evaporated water, which could not be replaced through the roots in the frozen ground.

Hosts:

Picea excelsa
Pinus sylvestris
P. nigra
P. contorta var. *latifolia*
P. montana
P. laricio var. *austriaca*
P. cembra
Pseudotsuga taxifolia (Kujala 1950)

Literature:

Gremmen, J. Some additional notes on *Crumenula* de Not. and *Scleroderris* de Not. Sydowia Ann. Mycol. 1955. 1955.

Kujala, V. Uber die Kleinpilze der Koniferen in Finnland. Commun. Inst. Forest. Fenniae 38 : 4. 1950.

Lagerberg, T. Granens topptorka. Medd. Stat. Skogsfors. anst. 10. 1913.

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DANGEROUS FOREST DISEASES IN NORTH AMERICA

Introduction

The North American report on native pathogens considered of potential danger to other countries has received the attention of the undersigned subcommittee of the IUFRO Working Group on International Cooperation in Forest Disease Research. Twenty-five forest tree diseases of serious incidence in North America were chosen for presentation at this Congress.

The subcommittee gratefully acknowledges the excellent cooperation of the various authors and the assistance of many colleagues in the United States and Canada who were so helpful in selecting and describing the diseases that are detailed on the pages that follow.

J. S. Boyce
J. R. Hansbrough
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Brooming Disease of Black Locust

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Chlorogenus robiniae Holmes. A systemic brooming virus of black locust in the United States. Brooms are found mainly on stump and root sprouts. There exist all degrees of brooming ranging from severe broom through mild broom to apparently healthy plants that are masked carriers of the infectious agent. Clearing of the leaflet veins is associated with early stages of the disease. There is a brooming of the roots as well as of the tops of the diseased plants. In midsummer brooms are usually found on the terminal growth of the leaders. Occasionally the brooming of terminal growth is followed by broom growth down the main stem and onto the side branches. Many broomed plants tend to lose their older leaves early. By late summer and early fall the diseased plants may have shed most of their lower leaves and succulent broom growth is very conspicuous. Herbarium specimens indicate that brooming has been present in some areas for more than 80 years.

Determination of the relative importance of the brooming disease is difficult because of the variable behavior of infected plants, some of which recover from symptoms whereas others die in part or as a whole. The virus can be carried in root cuttings and scion wood; thus intercontinental shipment of this type of propagating material should be avoided.

Range: United States from southern Pennsylvania to northeast Georgia and west to southwestern Ohio and Tennessee.

Hosts: Leguminosae; the brooming virus has been transmitted only by tissue grafts and only to *Robinia pseudoacacia* L. Similar brooming symptoms have been observed in a few instances on honey locust, *Gleditsia triacanthos* L.

Literature:

Grant, T. J., and Hartley, Carl. A witches'-broom on black locust and a similar disease on honey locust. Plant Dis. Rptr. 22: 28–31. 1938.

———, Stout, D. C., and Readey, J. C. A systemic brooming, a virus disease of black locust. Jour. Forestry 40: 253–260. 1942.

Holmes, F. O. Handbook of phytopathogenic viruses. Rockefeller Inst. for Med. Res., 221 pp. 1941. (Processed.)

Phloem Necrosis of Elm

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Morsus ulmi Holmes. An epiphytotic virus disease causing root rot, decline, and death of *Ulmus americana* and *Ulmus alata* in the United States. First symptoms are root rot and light-green, sparse foliage that becomes yellow; soon afterward followed by complete defoliation and death of the tree. The inner phloem tissue of large roots, stem, and some branches become discolored before their death; the discolored tissue is confined to the phloem and has a faint odor of wintergreen, or methyl salicylate. It is at first yellow, but the color changes to raw sienna and then to brown or black. The faint wintergreen odor can be detected most readily by confining some discolored phloem tissue in a stoppered vial for a few minutes. The raw sienna color and wintergreen odor of the inner phloem tissue are the specific symptoms of phloem necrosis that differentiate it from other known elm diseases.

The only known insect vector of the phloem necrosis virus is the leafhopper *Scaphoideus luteolus* Van D. The virus may be transmitted by budding or grafting, but mechanical inoculations with phloem and leaf extracts have not been successful. Infected trees do not usually develop typical disease symptoms until a year or more after inoculation. After initial disease symptoms appear trees usually die within 12 to 18 months, but some may die within 3 to 4 weeks.

Intercontinental spread may be possible through shipment of infected trees or scion wood, but the hazard of establishing the virus in new areas would depend on the occurrence of a suitable insect vector. The host range of the elm phloem necrosis virus is not well defined, and the possibility of importing plant species in which the virus may be latent exists.

Range: The central and plains States of the United States, bounded approximately N by 30 and 42 degrees latitude and W 80 and 100 degrees longitude.

Hosts: Ulmaceae—

Ulmus americana L.
Ulmus alata Michx.

Literature:

Anonymous. Control of Dutch elm disease and elm phloem necrosis. U.S. Dept. Agr. Leaflet 329: 1–11. 1952

Baker, W. L. Transmission by leafhoppers of the virus causing phloem nercosis of American elm. Science 108: 307–308. 1948.

Swingle, Roger U. Phloem necrosis. A virus disease of the American elm. U.S. Dept. Agr. Cir. 640: 1–8. 1942.

Atropellis Canker of Pine

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Atropellis piniphila (Weir) Lohman and Cash. A perennial canker on stems and branches of pines. The processes of disease development involving this pathogen have been examined in detail only on *Pinus contorta* in Alberta. The first symptom, consisting of a drop of resin on the surface of the bark, is frequently followed soon afterwards by rupture of the bark, revealing an underlying resin pocket. Fresh resin flow occurs early each summer from the canker margin throughout growth of most cankers.

The fungus invades all stem tissues, but grows more rapidly within the outer sapwood than within the bark. Growth is fairly slow in all directions, but proceeds much more rapidly in a longitudinal direction than in any other, resulting in a more or less diamond-shaped canker. Invasion inward within the wood proceeds primarily along the rays, but later spread laterally into the tracheids. The invaded wood is characterized by a blue-black discoloration, and frequently develops a peripheral incipient zone which possesses a reddish-brown color. Apothecia are formed on the surface of the bark at the canker centers, and formation continues at a distance of 6 to 9 inches from the longitudinal apices of the cankers.

The apothecia are erumpent, brownish to black, and 2 to 5 mm. in diameter when expanded by moistening. The clavate asci, interspersed with paraphyses, each contain eight hyaline, elliptical-fusoid ascospores, 16–22 × 4–5μ, which may be 1- or 2-celled before germination.

The disease is spread by ascospores, probably wind disseminated, which are released during wet weather in the growing season. Infection of the stems occurs mainly at the branch nodes. Girdling of the stem is very slow, but mortality may be produced in hitherto vigorous trees as a result of the occurrence of two or more cankers at the same level. Multiple stem cankers, common in high incidence areas, may cause considerable degrading of the wood for use as pulp or lumber.

Intercontinental spread possible, either through shipment of infected trees or infected bark on logs. Export logs should be debarked.

Range: This pathogen appears to form high incidence zones, mainly on *Pinus contorta* in Alberta and British Columbia. On this and other hosts, it occurs on a number of locations in western North America, including Montana, Idaho, Washington, Oregon, Arizona, and New Mexico; it has also been reported from Alabama and Tennessee.

Hosts: Pinaceae—
Pinus albicaulis Engelm.
P. contorta Dougl.
P. jeffreyi Grev. and Balf.
P. monticola Dougl.
P. ponderosa Laws.
P. taeda L.
P. virginiana Mill.

Literature:

Lohman, M. L., and Cash, E. K. *Atropellis* species from pine cankers in the United States. Jour. Wash. Acad. Sci. 30: 255–262. 1940.

Weir, J. R. *Cenangium piniphilum* N. sp., An undescribed canker forming fungus on *Pinus ponderosa* and *P. contorta*. Phytopathology 11: 294–296. 1921.

Weiss, F., and O'Brien, M. J. Pinaceae. *In* Index of plant diseases in the United States. Spec. Pub. 1, Pt. V: 827–850. U.S. Dept. Agr. Plant Dis. Surv. 1953.

Persimmon Wilt

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Cephalosporium diospyri Crandall. A vascular wilt of the genus *Diospyros* (mainly *D. virginiana* L.) in the United States. Symptoms are general wilting of the foliage followed by defoliation, brownish-black vertical streaking deeply through the sapwood of the trunk and branches, death of the tree within few months of first external symptoms, extensive fruiting of fungus as reddish blisters under firm bark or as large reddish powdery areas composed of enormous numbers of conidia exposed at the cambial region by exfoliating bark of recently killed trees. The most important features in diagnosis are sudden wilt; premature discoloration of foliage; heavy defoliation; streaking of wood not only in last annual ring but often through 10 or more rings of sapwood; simple isolation in agar culture, using sapwood chips, of *Cephalosporium diospyri* as described by Crandall (1945); powdery reddish spore masses in bark blisters or in masses under loose bark. *C. diospyri* differs from most other described members of the genus in its abundant production of orange-pink spores and in having a faint pink color in culture.

The causal fungus enters the vascular system through wounds, many of which are insect-caused. Spring infection results in rapid development of fungus through the vascular system, resulting in production of dark gums that produce the wood streaks, tyloses, and vessel plugging. Death commonly follows in 2 to 3 months, with sporulation usually following immediately, from August to late autumn.

Range: This disease killed on a catastrophic scale in central Tennessee and little persimmon remains in that area. It has also caused varying losses in an area embraced by the northern boundaries of North Carolina and Tennessee southward to the Gulf of Mexico, extending through the north half of Florida, and west to the

Mississippi River, with a few infections in Texas. A large part of the persimmon range, such as the main Mississippi River Delta, the Appalachian Mountain chain to Georgia and Alabama, and the area north and west of North Carolina and Tennessee has remained free of wilt so far as is known. The disease was especially severe in the period 1930–50, and while occasional cases are still reported in the eastern States, and there have been recent outbreaks in Texas and Oklahoma, it appears much less commonly now than in the 1930's and still has not invaded many areas where persimmon abounds.

Hosts: Ebenaceae—

Diospyros virginiana L., very susceptible
D. ebenaster Ratz, very susceptible
D. texana Scheele, susceptible
D. lotus L., resistant
D. kaki L., resistant
D. rosei Standley, immune
D. mosieri Small, untested, but wilt-free in Florida

Literature:

Crandall, Bowen S. Spread of persimmon wilt. Plant Dis. Rptr. 27: 158–160. 1943.

———. A new species of Cephalosporium causing persimmon wilt. Mycologia 37: 495–498. 1945.

——— and Baker, W. L. The wilt disease of American persimmon caused by Cephalosporium diospyri. Phytopathology 40: 307–325. 1950.

Toole, E. R., and Lightle, P. C. Status of persimmon wilt, 1959. Plant Dis. Rptr. 44: 45. 1960.

Oak Wilt

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Ceratocystis fagacearum (Bretz) Hunt. A vascular wilt disease of oaks, known only in the eastern half of the United States. Symtoms differ between species of the red oak group and those of the white oak group. In red oaks, symptoms are characterized by a wilting and bronzing of the foliage, starting at the top and tips of branches and spreading rapidly throughout the entire crown. Individual leaves bronze progressively from the tip to the base, oftentimes leaving a localized area of green tissue at the base around the midrib. Leaves in all stages of discloroation, including green leaves, are shed more or less continuously as the disease progresses. Bole sprouting is often observed on wilting trees.

Trees of all ages are affected and usually die the same season symtoms appear. Occasionally large trees, or trees affected late in the season, survive until the following spring, but, invariably, all infected red oaks die. White oaks are generally more resistant to the disease, however, and may live several or more years before they die. In some cases, infected white oaks recover; diseased trees showing no foliar symptoms have been reported. Affected trees usually exhibit symptoms in one branch at one time as the disease slowly progresses through-

out the crown. Generally, bronzing is less pronounced and defoliation is reduced in diseased white oaks. Streaking commonly occurs in the outer sapwood vessels of white oaks, but is less frequently found in red oaks.

The oak wilt fungus produces abundant endoconidia in gray or tan mats formed beneath the bark of infected dying or dead trees. Mat production varies widely within the range of the disease and is more abundant on red than on white oaks. Endoconidia are 1-celled, hyaline, cylindrical, 2–4.5 × 4–22μ, and are produced in slightly tapering hyaline to brown conidiophores. Cushion-shaped structures, called pressure pads, are usually formed in mat centers and exert sufficient pressure to raise and often split the bark, exposing the fruiting surface of the mat.

Since this fungus is heterothallic, perithecia are produced only when exposed mats are spermatized with an opposite compatibility type. Spermatization is accomplished by insects which happen to visit both types and accidentally transfer conidia of one type to the mat of another. Both compatibility types, A and B, are common in nature, but do not often occur in the same tree.

The principal features of the perfect stage, adapted from the original description are as follows: Perithecia flask-shaped, black with spheroidal base, 240–380μ in diameter, largely embedded in the subiculum, appearing in 7 to 10 days in culture inoculated with compatible isolates; walls membranous to leathery; usually 1, rarely 2, erect beaks, 250–450μ long, black, terminated by a fringe of hyaline filaments; asci 8-spored, evanescent; ascospores hyaline, 1-celled, elliptical and slightly curved, 2–3 × 5–10μ, collecting at the ostiole in a sticky, creamy-white mass; hermaphroditic, self-sterile but cross-fertile.

Approximately one out of every two infections spreads locally, while the remaining infections, usually single trees, die without further spread. Some of the local spread is known to occur through root grafts connecting diseased and healthy trees. Other local spread, as well as long distance spread, occurs overland. Overland spread probably takes place by means of insects, but evidence is lacking on the relative importance of specific vectors. However, the mats produced on wilt-killed trees have a fruity odor and are attractive to insects.

Sap-feeding nitidulid beetles, which commonly visit these trees, become heavily contaminated with spores and can transmit the fungus when they feed on the sap of fresh wounds on healthy trees. Also, bark beetles (*Pseudopityophthorus* spp.) and borers (*Agrilus bilineatus* and *Graphisurus fasciatus*) reared on wilt-killed trees on which no mats occurred were found to be contaminated with spores. In limited tests under caged conditions, contaminated bark beetles were further shown to transmit the disease to healthy trees through their feeding wounds. However, the role these insects play in the natural spread of the disease is, as yet, unknown. The fungus does not appear to be ideally suited to wind dissemination.

Based on the slow rate of spread and the generally negligible losses experienced since the disease was identified almost 20 years ago, there is hope that the disease will never develop epiphytotic proportions. Nevertheless, oak wilt is well established in the natural range of oak, and has caused extensive damage in a few localized areas. Furthermore, oaks are species of high value, comprising one-third of

the hardwood sawtimber. For these reasons, and because the disease is usually lethal, oak wilt must be considered a serious threat to the hardwood industry.

Statewide control programs, aimed at eliminating the source of inoculum, are active in a number of States. Principal suppression measures include (1) the felling of diseased trees and all healthy oaks within possible root graft distance (about 50 feet) and poisoning the stumps; (2) felling diseased trees only, poisoning stumps, and spraying infected trees and stumps with No. 2 fuel oil containing DDT, BHC, and pentachlorophenol; and (3) deep girdling infected trees to the heartwood to hasten drying and inhibit mat production. While it is expected that these measures help to suppress oak wilt, their effectiveness has not been clearly demonstrated.

Intercontinental spread may be possible through the shipment of logs or lumber cut from infected trees. As a result, several countries have placed embargoes on oak logs and lumber from the United States unless certified disease-free. Oak lumber may be certified if kiln-dried to a moisture content of 20 percent or less, and both logs and lumber may be certified if originating outside the disease area. Certificates are required for shipments to Egypt, Germany, Italy, Sweden, Turkey, Union of South Africa, Yugoslavia, and possibly, also Portugal.

Range: Oak wilt is widespread throughout the eastern and midwestern United States, occurring in 19 States, from Nebraska and Oklahoma in the West to Pennsylvania and North Carolina in the East.

Hosts: Fagaceae. Susceptible species indigenous to the United States found naturally infected include *Quercus alba*, *Q. bicolor*, *Q. coccinea*, *Q. ellipsoidalis*, *Q. falcata*, *Q. ilidifolia*, *Q. imbricaria*, *Q. macrocarpa*, *Q. marilandica*, *Q. muehlenbergii*, *Q. palustris*, *Q. phellos*, *Q. prinus*, *Q. rubra*, *Q. stellata*, and *Q. velutina*. Chinese chestnut, *Castanea mollissima*, an exotic, was also found to be susceptible through natural infections.

Additional species, both native and exotic, which have been shown to be susceptible through artificial inoculation include *Quercus falcata* var. *pagodaefolia*, *Q. michauxii*, *Q. nigra*, *Q. shumardii*, *Q. agrifolia*, *Q. chrysolepis*, *Q. gambelii*, *Q. garryana*, *Q. laevis*, *Q. laurifolia*, *Q. lobata*, *Q. shumardii* var. *texana*, *Q. virginiana*, *Q. virginiana* var. *maritima*, *Q. wislizenii*, *Q. robur*, *Q. acutissima*, *Q. acuta*, *Q. aliena accutesserata*, *Q. brutia*, *Q. castanaefolia*, *Q. cerris*, *Q. dentata*, *Q. gardeniaefolia*, *Q. glandulifera*, *Q. glauca*, *Q. haas*, *Q. ilex*, *Q. longiux*, *Q. lusitanica*, *Q. macrolepis*, *Q. myrsinaefolia*, *Q. suber*, *Q. thomasi*, *Q. variabilis*, *Castanea dentata*, *C. sativa*, *C. pumila*, *Castanopsis kawakamii*, *C. sempervirens*, *Lithocarpus glabra*, *L. densiflorus*.

Literature:

Bretz, T. W. The ascigerous stage of the oak wilt fungus. Phytopathology 42: 435–437. 1952.

Henry, B. W. *Chalara quercina* n. sp., the cause of oak wilt. Phytopathology 34: 631–635. 1944.

True, R. P., Barnett, H. L., Dorsey, C. K., and Leach, J. G. Oak wilt in West Virginia. W. Va. Univ. Agr. Expt. Sta. Bul. 448T: 1–119. 1960.

Canker Stain of Plane Trees

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Ceratocystis fimbriata Ell. & Halst. (*Endoconidiophora fimbriata* (Ell. & Halst.) Davidson f. *platani* Walter). A lethal bark canker and wood-stain of plane trees in the United States. London plane (*Platanus acerifolia* Willd.), extensively planted as a street tree in the United States, is killed by the disease and *Platanus occidentalis* L. is damaged but rarely killed. Cankers on young, yellow or green bark are at first slightly sunken, lens-shaped, or elongated areas in line with the grain of the underlying wood. Older cankers are sunken, elongate, irregular in outline, becoming rough and blackened. Infected bark dries, cracks, and falls. Wood is discolored reddish brown or bluish black, especially the medullary rays. In cross section, discolored areas of wood often are wedge-shaped. Cankers eventually coalesce. Limbs and trunk are girdled; members distal to the cankers die.

The first leaf symptoms to appear are usually dwarfing and yellowing. Leaves eventually wilt and fall in summer. On small branches or small stems the disease may cause wilting and defoliation of normal-size green leaves. By the time leaf symptoms become evident, canker development and invasion of wood are extensive.

Canker stain is the most serious disease of *Platanus acerifolia* in the United States. Although canker stain is most destructive on the introduced London plane, the disease has been observed on the native *P. occidentalis* in wild areas far from cultivated London plane trees. The causal fungus is readily transmitted on prunning tools of all kinds. The fungus invades through wounds. Natural spread occurs but does not appear to be important in comparison with spread through use of contaminated pruning tools and contaminated, non-antiseptic tree wound paints. In limited tests, isolates of the fungus from London plane were not pathogenic to sweet potato or to cacao seedlings.

Conidia and perithecia containing ascospores are produced on cankers. Thick-walled, brown spores form in invaded wood. All spore types can infect.

The fungus is indistinguishable morphologically from *Ceratocystis fimbriata.* Ascospores 4.5–8 × 2.5–5μ. Endoconidia 11–16 × 4.5μ.

Range: Eastern United States

Hosts: Platanaceae—

Platanus acerifolia Willd.
P. occidentalis L.
P. orientalis L.

Literature:

Hunt, J. Taxonomy of the genus *Ceratocystis.* Lloydia 19: 1–59. March 1956.

Walter, J. M. Canker stain of plane trees. U.S. Dept. Agr. Yearbook 1943–47: 481–484. 1947.

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Clitocybe Root Rot

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Clitocybe tabescens (Fr.) Bres. A destructive disease of fruit, forest, shade and ornamental trees, shrubs, and vines mainly in the Southeastern United States. Symptoms vary with the kind and size of the host and the rapidity with which the fungus girdles the affected plant. Sudden wilting is often the first symptom in broadleaf trees; however, yellowing, partial defoliation, and a general unthrifty appearance are common top symptoms where girdling proceeds more slowly. By the time top symptoms are evident, mycelial growth of the fungus has progressed from the roots into the base of the tree causing slightly sunken lesions which may extend upward from a few inches to a foot or more above ground. Extensive mycelial fans develop under the basal bark lesions and under the bark on diseased roots. Mycelial mats vary from thin filmy wefts to leathery sheets that are white when fresh but which become cream to chamois colored with age.

Fruiting bodies of the gill fungus *Clitocybe tabescens* may develop at the base of affected plants depending upon the progress of the disease and seasonal conditions. These may be sparse or abundant in late summer and fall in the Southern United States, depending on moisture and temperature. The fruiting bodies usually consist of few to many individuals with the stems developing from a common base. An annulus is lacking. When fully developed the caps are convex to flattened or centrally depressed with age, whitish to light tan or honey colored, smooth or with tufts of fibrils near the center and from 5 to 9 centimeters in diameter with whitish gills.

Bresadola considers the American plant synonymous with *Clitocybe tabescens* of Europe, however, judging from the lack of references in the literature it causes little damage there. Root rot caused by *C. tabescens* is similar to that caused by the closely related *Armillaria mellea* Fr. and losses by these two fungi are probably confused in the literature. Clitocybe root rot has been identified as the more serious cause of mortaility in cultivated trees and shrubs.

Range: In the United States mainly from eastern Texas, Oklahoma, Missouri, southern Illinois, and Indiana, West Virginia, Virginia southeastward to Florida. The fungus occurs north and west of this range but rarely associated with root rot. It has been reported from Madagascar and India.

Hosts: In Florida Clitocybe root rot has been recorded on 210 species of plants belonging to 137 genera and 59 families, including Pinaceae, Cupressaceae, Casuarinaceae, Fagaceae, and Myrtaceae.

Literature:

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Spruce Broom Rust

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Chrysomyxa arctostaphyli Diet. (*Peridermium coloradense* A. & K.) is a perennial witches'-broom of spruce in North America. First symptom is probably needle etiolation. Release of dormant buds results in formation of brooms, which bear annual crops of yellow-green needles. Abundant pycnia produced on these needles in late spring give off a strong, foul odor detectable for 50 meters or more. Aeciospores produced during summer give entire brooms a yellow-orange appearance. Fungus mycelium invades bark and outer xylem of branches and trunks, often giving rise to secondary brooms and occasionally causing formation of cankers and fusiform swellings.

Aeciospores infect *Arctostaphylos uva-ursi* (L.) Spreng., a common associate of spruce in Eurasia. Basidiospores produced on this trailing, woody plant are believed to infect spruce in early summer. There are no urediniospores. It has been suggested, but not confirmed, that spruce-to-spruce transmission by aeciospores also occurs.

Spruce broom rust is similar to the broom rust of *Abies* (*Melampsorella caryophyllacearum* (DC.) Schroet.). They may be distinguished not only by host but also by the looser, larger brooms on spruce and by microscopic characteristics of the fungi. Distinct woody swellings are more commonly caused by the fir parasite, which also changes the shape and color of infected needles more than does the spruce rust.

Spike-tops, dead branches, and mortality, are commonly associated with spruce brooms. Presence of dead brooms near trunks is usually an indicator of heart rot.

Seldom are more than 25 percent of the spruces in a stand infected: in most stands fewer than 1 percent bear brooms.

Intercontinental spread is possible on infected plants and perhaps by windblown aeciospores, which survive several months' storage. However, the telial host is a very unlikely target because of its thick cuticle and lack of stomata on upper leaf surfaces.

Range: Nearly coextensive with the range of spruce in North America, but more common in the West.

Hosts:

Pinaceae—
- *Picea abies* (L.) Karst. where planted
- *P. engelmannii* Parry
- *P. glauca* (Muench) Voss
- *P. mariana* (Mill.) BSP
- *P. pungens* (Engelm.)
- *P. rubens* Sarg.
- *P. sitchensis* (Bong.) Carr.

Ericaceae—
- *Arctostaphylos uva-ursi* (L.) Spreng.

Literature:

Bourchier, R. J. Alberta and Rocky Mountain Parks forest disease survey. Forest Ins. and Dis. Surv., Div. For. Biol., Sci. Serv., Canad. Dept. Agr., Ann. Rpt. 1952: 121–126. 1953.

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Comandra Blister Rust

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Cronartium comandrae Pk. (Syn.: *C. pyriforme* Hedg. & Long) is a disease of hard pines in North America. It is perennial in the living bark of the pine hosts: develops annually on false toadflaxes, the alternate hosts. First conspicuous symptom of the disease on pines is a spindle-shaped swelling of the bark on branches and on stems of small trees. Cankered bark is usually constricted on main stem or trunk of large or mature trees. Generally, there is copious resin exudation from trunk cankers, especially in *Pinus contorta.* Rodents, mainly porcupines and squirrels, often feed on the infected bark. Wounds inflicted by them may increase resin flow. Dead and dying tops and branches are conspicuous symptoms of this rust in invaded pine stands. Cankers originate on needle-bearing twigs and stems.

Large, reddish-orange pycnial drops (4–8 mm. diam.) appear on swollen bark 2 to 3 years after initial infection. Aecia are produced the following spring and summer. Aeciospores pyriform; this character readily separates this blister rust from all others on pines. Tiny pycniospores have the same general shape. The life cycle and character of attack on pines are very similar to that of the well-known white pine blister rust (*C. ribicola* Fischer). Aeciospores are wind disseminated to alternate host plants. Uredia appear about 10 days after infection; telia begin to develop about 15 days later. Airborne basidiospores infect pines in late summer and fall, thus completing the life cycle.

Comandra blister rust is a very destructive disease in Western United States. *Pinus contorta*, *P. ponderosa*, and *P. pungens* are highly susceptible to the fungus. The degree of susceptibility of other reported pine hosts is not precisely known. Infected trees are killed by girdling. Seedlings are killed in a few years; 25 years or more may elapse between initial infection and death of mature pines.

No method of control has yet been developed. Grubbing out of alternate host plants is not feasible as a control measure. Herbicides are not effective on *Comandra umbellata*, a xerophilous plant inhabiting open rangeland and of practically no forage value. *C. livida* occurs on wet sites; rare in U.S., but fairly common in Canada. *Buchleya distichophylla* (Nutt.) Torr., was erroneously reported as an alternate host by J. C. Arthur.

Range: North America. New Brunswick to the Yukon and British Columbia in Canada and southward to northern Mississippi, New Mexico, and California in U.S.

Hosts:

Pinaceae—
Pinus banksiana Lamb
P. contorta Dougl.
P. nigra Arnold (Planted in U.S.)
P. pinaster Aiton (Planted in U.S.)
P. ponderosa Laws.
P. ponderosa var. *arizonica* (Engelm.) Shaw
P. pungens Lamb
P. rigida Mill.
P. sylvestris L. (Planted in U.S.)
P. taeda L.

Santalaceae—
Comandra umbellata (L.) Nutt., syn. (*C. pallida* A. DC.)
C. livida Richards (*Geocaulon lividum* (Richards) Kern.)

Literature:

Arthur, J. C. Manual of the rusts in the United States and Canada. Lafayette: Purdue Res. Found., 438 pp. 1934.

Hedgcock, C. G., and Long, W. H. A disease of pines caused by *Cronartium pyriforme*. U.S. Dept. of Agr. Bul. 247, 20 pp. 1915.

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Southern Fusiform Rust

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Cronartium fusiforme Hedgc. & Hunt is a heteroecious long-cycle rust causing perennial stem and branch galls on *Pinus* and leaf spots on *Quercus* in Southeastern United States. Bark swellings usually are first evident in early fall following spring infection, but sometimes are not pronounced until second year. Galls are typically spindle shaped, may finally reach a meter or more in length, and on large boles usually become sunken cankers with age. Proximal growth of branch galls is often irregular so that only the distal end may be fusiform. Rate of gall elongation is very variable but averages 75 to 125 mm. per annum.

Aecial fruiting biennial, bright yellow orange, February to April; peridium erumpent, cerebroid, moderately thin (1 to 2 cells). Aeciospores 13–18 × 22–28μ, coarsely verrucose. Following aeciospore infection of oaks, pale green leaf spots develop, quickly followed by bright orange uredial pustules on the under surface in February to May. Uredospores 12–15 × 17–21μ, evenly and sharply echinulate. Telia appear in uredinial spots or, more frequently, independently from February to June; are brown, average 104 × 2.872μ, with teliospores averaging 14.7 × 36.4μ. Pycnia appear in October to April; pycnial ooze at first pale orange but soon becomes a black splotch on the bark. Spores are windborne and pine infection occurs through first-year needles.

This is one of the most serious diseases of *Pinus taeda* and *P. elliottii* var. *elliottii*. Pines of all ages are susceptible, but most

damaging infections occur on seedlings less than 4 years old. Early infections usually lead to death, and later infections to deformities and wind breakage. Without fungicidal control, losses in nurseries are commonly 10 to 20 percent and sometimes 50 to 90 percent. Damage to oak is minor.

Intercontinental spread is possible through the shipment of infected pines. Some infection is latent until the second year. There appears very little chance of spread by the shipment of seed or pollen. Because infections on oak are restricted to leaves there should be no risk in shipment of dormant oak seedlings or of acorns.

Range: United States. Maryland to Florida and westward to Arkansas and Texas. Common in the coastal States from South Carolina to Texas.

Hosts:

Pinaceae (aecial and pycnial stages)—
Pinus elliottii var. *densa* Little & Dorman
P. elliottii var. *elliottii* Little & Dorman
P. palustris Mill.
P. rigida Mill.
P. serotina Michx.
P. taeda L.
P. caribaea Morelet (Planted in U.S.)
P. cooperi var. *ornelasi* Martinez (Planted in U.S.)
P. nigra Arnold (Planted in U.S.)
P. pseudostrobus Lindl. (Planted in U.S.)
P. torreyana Parry (Planted in Southeast U.S.)

Artificial inoculations proved 13 other *Pinus* species are susceptible, among them *P. radiata* Don and *P. sylvestris* L.

Fagaceae (uredial and telial stages), with first 8 in order of susceptibility—
Quercus nigra L.
Q. phellos L.
Q. laurifolia Michx.
Q. incana Bartr.
Q. marilandica Muenchh.
Q. falcata Michx.
Q. laevis Walt.
Q. virginiana Mill.
Q. alba L.
Q. coccinea Muenchh.
Q. imbricaria Michx.
Q. shumardii Buckl.
Q. stellata Wangenh.
Q. velutina Lam.

Artificial inoculations have proved many other oak species susceptible, including *Q. robur* L. and *Q. cerris* L.; 4 species of *Castanea* (*dentata* (Marsh.) Borkh., *mollissima* Bl., *pumila* Mill., and *sativa* Mill.); *Castanopsis diversifolia* (Kurz) King; and *Lithocarpus densiflora* (Hook and Arn.) Rehd.

Literature:

Hedgcock, G. G., and Siggers, P. V. A comparison of the pine-oak rusts. U.S. Dept. Agr. Tech. Bul. 978: 1–30. 1949.

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Eastern Gall Rust

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Cronartium quercuum (Berk.) Miyabe ex Shirai is a heteroecious rust, the aecial stage of which induces perennial globose galls on stems and branches of two- and three-needle pines in eastern North America. Initial symptoms on pines are a slight hemispherical swelling on one side of the stem usually in the region of the previous season's growth. This swelling enlarges, becomes spherical, and often reaches twice the diameter of the normal stem by the second year. Advance is more rapid in the tangential direction than in the longitudinal. In a few years the gall may completely girdle the stem, but frequently it persists for many years, continuing to enlarge without girdling, and causing a reduction in tree vigor. The soft wood of the gall is liable to insect and rodent attack which results in entry courts for decay fungi and susceptibility to wind breakage.

Droplets of the sticky viscous pycnial fluid borne in lenticular cavities in the gall epidermis may appear the first, but more commonly the second or third spring after infection. Pycniospores are hyaline, oblong to elliptic, approximately 2 × 4μ. Aecia, with thick cerebroid peridia, appear a year later on the same gall. Aeciospores are yellow orange, obovoid or ellipsoid, wall coarsely verricose with one flat side, 15–18 × 24–29μ.

The uredinial and telial stages occur annually on the undersurface of leaves of oak and chestnut. Urediospores are yellow, ovoid to ellipsoid, wall echinulate, 11–15 × 18–24μ. Chestnut-brown telial columns appear in the same or new sori after the uredia. Basidiospores are hyaline, ellipsoid, approximately 4 × 6μ. Where the ranges overlap, this rust is difficult to distinguish from several other pine-oak rusts on the oak leaves.

In some regions this is a serious disease of pine in nurseries and young plantations, although the incidence of new infection fluctuates from year to year. Trees are most susceptible in the seedling stage. Infections on the main stem usually result in mortality or severe stunting.

Intercontinental spread is most probable through shipments of seedlings. The risk is increased by the fact that symptoms might not appear for 1 to 3 years. Consequently, importations should be kept under close surveillance for several years after planting.

The taxonomy of this rust is uncertain. Some workers believed it to be distinct from the Asiatic form. However, the above name, based on the Asiatic Type Collection, is the only valid name in accordance with the International Code. In view of this confusion, importation of susceptible hosts into Asia should be made with as much caution as into other continents.

The fungus overwinters as mycelium in the pine gall. Pycnia are are produced in early spring. Aecia appear beneath the pycnial scars one year later. Rupture of the peridia and wind dissemination of

the aeciospores usually coincides with the bud break of the oak. Uredia appear on the oak leaves 1 to 2 weeks after infection by aeciospores. Urediospores may repeat infections on oak until leaves become immune, 2 to 3 weeks after they are fully expanded. Telia appear in early summer and produce basidiospores which infect the young growth of the pine from early to midsummer.

Range: North America. Common from Maine, Ontario, and Minnesota to Florida and Texas.

Hosts:

Pinaceae (0) (I)—
- *Pinus banksiana* Lamb.
- *P. clausa* (Engelm.) Vasey
- *P. echinata* Mill.
- *P. elliottii* Engelm.
- *P. glabra* Walt.
- *P. nigra* Arnold (Planted in U.S.)
- *P. ponderosa* Laws.
- *P. pungens* Lamb.
- *P. resinosa* Ait.
- *P. rigida* Mill.
- *P. sylvestris* L. (Planted in U.S.)
- *P. taeda* L.
- *P. thunbergii* Parl. (Planted in U.S.)
- *P. virginiana* Mill.

Fagaceae (II) (III)—
- *Castanea dentata* (Marsh.) Borkh.
- *C. pumila* (L.) Mill.
- *Quercus alba* L.
- *Q. bicolor* Willd.
- *Q. coccinea* Muench.
- *Q. ilicifolia* Wangenh.
- *Q. imbricaria* Michx.
- *Q. macrocarpa* Michx.
- *Q. marilandica* Muench.
- *Q. myrtifolia* Willd.
- *Q. nigra* L.
- *Q. palustris* Muench.
- *Q. phellos* L.
- *Q. prinus* L.
- *Q. rubra* L.
- *Q. stellata* Wangenh.
- *Q. velutina* Lam.
- *Q. virginiana* var. *minima* (Small) Sarg.

Literature:

Cummins, G. B., and Stevenson, J. A. A check list of North American rust fungi (Uredinales). Plant Dis. Rptr. Sup. 240: 109–193. 1956.

Dodge, B. O., and Adams, J. F. Some observations on the development of *Peridermium cerebrum.* Torrey Bot. Club Mem. 17: 253–261. 1918.

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Ponderosa Pine Needle Cast

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Elytroderma deformans (Weir) Darker. A perennial disease of pitch pines in North America. Infected needles are usually stunted, becoming pale near the end of their first summer and red brown the following spring, fading to gray in the late summer and falling in the early winter of their second year. Necrotic lesions form in twig phloem, tips of twigs curl upward, and old infections on trees of fair to good vigor develop into compact, globose witches'-brooms. On year-old needles, inconspicuous tendrils of hyaline conidia appear in the spring; dark, linear hysterothecia become conspicuous during the summer. Hysterothecia are amphigenous, sometimes several mm. long, scattered or in series, and opening by a longitudinal fissure; asci are fusiform and 30–45 × 140–240μ; paraphyses, filiform and 2- or 3-septate; ascospores are 1-septate at maturity, cylindric or slightly fusiform, often somewhat curved 6–8 × 90–118μ, surrounded by a gelatinous sheath, and discharged in the fall.

Requirements for infection by spores are apparently exacting, but heavy waves of infection sometimes occur on trees of all sizes, causing severe local damage. The fungus perennates in twig phloem and buds, invading needle primordia soon after their formation. If most of the twigs on a tree become infected, recurrent annual infection by spores is not necessary for death to occur in 4 or 5 years from defoliation. Less severe infection retards growth and deforms young trees.

Intercontinental spread is possible through shipment of infected trees. Retention in quarantine is *not* a sufficient safeguard, since diseased twigs occasionally become symptomless for a year or longer and then once again produce fruiting bodies of the fungus on their needles.

Range: In North America it is common from western Canada southward into California and eastward to the Rocky Mountains; rare in eastern Canada and United States.

Hosts: Pinaceae—

Pinus ponderosa Laws.
P. jeffreyi Grev. & Balf.
P. contorta Dougl.
P. edulis Engelm.
P. banksiana Lamb.
P. echinata Mill.

Literature:

Darker, G. D. The Hypodermataceae of conifers. Contrib. Arnold Arboretum I: 1–131, illus. 1932.

Weir, J. R. Hypoderma deformans, an undescribed needle fungus of western yellow pine. Jour. Agr. Res. 6: 277–288, illus. 1916.

Gray Blight of Hard Pines

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Hypoderma lethale Dearness is a needle blight and cast of hard pines in the eastern United States. The first symptoms occur on the youngest needles as a rather sudden discoloration in late winter or early spring before new needle growth begins. In early spring, the initial zones of infection become reddish brown in color and are often separated by greenish areas of the leaf or they may be terminal. About this time of year the diseased portions may become slightly wrinkled and pycnidia, concolorous with the needle, develop on them. In late spring the initial areas of infection usually become straw colored or grayish and hysterothecia begin to appear within them. These areas often remain bounded by darker orange-brown zones while the green portions between infected areas turn brown.

The elliptical, shining black, flat, subepidermal hysterothecia mature in late spring or early summer and are 0.4–1.4 mm. long, 0.18–0.42 mm. wide, and 0.13–0.21 mm. deep. The hysterothecia open narrowly by a longitudinal fissue exposing the concolorus hymenium composed of narrow, filiform paraphyses and 8-spored asci, the latter 90–160 × 16–22μ. The ascospores are short, bacillar to fusiform, hyaline, 24–40 × 3–6μ, and are surrounded by a gelatinous sheath; on germination they divide and there emerges from one cell a short germ tube from which a terminal swollen portion is delimited by a septum. Infection is by windborne ascospores and takes place during the period of ascospore discharge and germination, principally during June and July in the more southern States and perhaps extending into August in the more northern areas.

This is one of the most serious and widespread needle diseases of hard pines in the eastern United States. Trees ranging from seedlings to sawlog size are infected. Destruction of 40 percent of the needle tissue per tree has been reported although at present direct evidence of significant reduction of the growth of infected trees is still lacking.

Intercontinental spread of the disease could most possibly come about through shipment of seedlings, especially in the late fall when the symptoms are latent. Rigorous inspection in the late spring prior to needle emergence should reveal the presence of the fungus and provide a sounder basis for prevention of its spread into new areas. Control of the disease by spraying, though possible, does not at present appear to be feasible on a large scale. *Pezizella minuta* Dearness, a secondary fungus, which fruits only on needles infected by *Hypoderma lethale* and prevents the maturation of the latter, provides a limited natural control and its importation into infected stands would no doubt have some beneficial effect.

Range: Entire eastern seaboard of the United States (except Delaware); along the Gulf of Mexico west to Louisiana; and scattered stations in Tennessee, West Virginia, Ohio, Michigan (?), and Missouri. Perhaps more widely spread throughout the eastern United States and adjacent regions than present knowledge indicates.

Hosts: Pinaceae—

Pinus clausa (Engelm.) Vasey. Florida.
P. echinata Mill. Pennsylvania, Virginia to Florida, Alabama, Louisiana, West Virginia, Tennessee, Missouri.
P. elliottii Engelm. var. *elliottii*. South Carolina to Florida, Mississippi.
P. nigra Arnold. New York, Pennsylvania.
P. nigra var. *austriaca* Aschers. & Graebn. Rhode Island, New Jersey.
P. pungens Lamb. North Carolina.
P. resinosa Ait. New York (?), Michigan (?).
P. rigida Mill. Maine to South Carolina, Mississippi, Ohio, Tennessee.
P. serotina Michx. North Carolina to Florida.
P. taeda L. Virginia to Florida, Alabama, Tennessee.
P. virginiana Mill. Pennsylvania to Florida, Tennessee.

Literature:

Boyce, J. S., Jr. Hypoderma needle blight of southern pines. Jour. Forestry 52: 496–498. 1954.
Darker, G. D. The Hypodermataceae of conifers. Contrib. Arnold Arboretum I: 1–131. 1932.
Hedgcock, G. G. Notes on the distribution of some fungi associated with diseases of conifers. Plant Dis. Rptr. 16: 28–42. 1932.
Morris, C. L. Chemical control of Hypoderma lethale on pitch pine. Plant Dis. Rptr. 37: 368–370. 1953.

Hypoxylon Canker of Aspen

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Hypoxylon pruinatum (Klotsche) Cooke is a perennial, girdling, stem and branch canker of *Populus* in North America. The first symptom on aspen bark is small, yellowish-orange, slightly depressed area with irregular margin. Later, the outer bark is raised in blister-like patches, sloughs off, and exposes the blackened, crumbling inner bark. Cutting into the bark of young cankers or near the margin of older cankers shows the laminated, mottled, black and yellowish-white cortex. Removal of all the bark exposes the typical white mycelial fans in the cambial zone. Advancing canker margin is irregular, yellowish orange, sometimes marked by a brownish varnishlike sap flow. Invasion of new tissue is too rapid to permit callus formation.

Conidia are produced under blisters at the end of the first or during the second year after infection, accompanied by formation of hyphal pegs or pillarlike structures that push the outer periderm from the underlying cortical tissue. Conidia are one-celled, oblong to ovoid, hyaline (brownish in mass), 4–7 × 1–2μ. In the third year perithecia are produced in erumpent, flattened, hard, black stromata, thinly covered with a white pruinose layer through which protrude the black ostioles of the sunken perithecia. Asci are cylindrical, with eight one-celled, brown, oblong to elliptical ascospores, 22–30 × 8–13μ.

This is the most serious disease of the aspens in North America. Trees of all ages are susceptible. Infection is by windborne spores

through bark wounds. Infected trees are killed by girdling or by stem breakage at cankers. Losses are heavy, up to 70 percent mortality in some stands.

Intercontinental spread is possible through shipment of infected trees or cuttings or of infected bark on logs or lumber. Importation of living plant material other than pollen or seed should be forbidden except for experimental use following rigorous inspection and retention in quarantine until all latent infections have had time to appear. Export logs and lumber should be debarked.

Range: In North America it is common from eastern Canada south to Virginia and west to the Great Plains; scattered in western United States and Canada.

Hosts: Salicaceae (species listed in order of susceptibility)—
Populus tremuloides Michx.
P. grandidentata Michx.
P. balsamifera L.
P. tremula L. (planted in U.S.)
P. adenopoda Maxim. (planted in U.S.)
P. alba bolleana Lauche (planted in U.S.)

Literature:

Bier, J. E. Studies in forest pathology. III. Hypoxylon canker of poplar. Canad. Dept. Agr. Tech. Bul. 27 : 1–40. 1940.

Gruenhagen, R. H. Hypoxylon pruinatum and its pathogenesis on poplar. Phytopathology 35 : 72–89. 1945.

Cedar Leaf Blight

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Keithia thujina Durand is a leaf disease of western red cedar in North America. Worse in localities of high atmospheric humidity. The lower branches of young trees, when growing in dense stands, often appear at a distance as if scorched by fire. Foliage of the upper crowns of mature trees may be generally infected but never to the same degree as leaves on lower branches near the ground. In late autumn the young infected leaf twigs drop, leaving the branches somewhat bare. On leaves remaining attached to the older twigs the fructifications drop out leaving deep pits. The fructifications (apothecia) are usually on the upper surface of the leaves, although they occasionally occur on the undersurface They are embedded in the leaf tissue and are exposed by the rupture of the epidermis in a flap- or scale-like manner. The apothecia are cushionlike, depressed when the air is dry, elevated when it is moist, and are circular, elliptical, curved or irregular in outline. The apothecia are olive brown at first but, with age, become almost black. Each ascus has two hyaline elliptical or pyriform spores, which when mature become olive brown and nearly circular in shape. The spores are pitted and very unequally two-celled. The paraphyses are club-shaped and single or branched. They have cross walls. No conidial stage is known.

This is a severe foliage disease of western red cedar in North America. Primarily a disease of seedlings and saplings, trees less

than 4 years old may be killed in one season, whereas on young trees beyond the seedling stage the effect is severely retarded growth. Infection of the leaves is by windborne spores.

The fungus is present in Great Britain, Ireland, and western Europe. The manner of its introduction is unknown but it probably came in on living plants, although it has been suggested that it came on or with seed. Living material of western red and northern white cedars should not be moved from region to region.

Range: In North America it is prevalent from Alaska southward along the coast ranges of British Columbia, western Washington and Oregon, eastward to Idaho, Montana, and eastern British Columbia. In the eastern United States it is recorded in the Lake States and Vermont.

Hosts: Cupressaceae—

Thuja plicata D. Don (western red cedar)

T. occidentalis L. (northern white cedar)

Literature:

Boyce, J. S. Forest pathology. Ed. 2, 550 pp. New York: McGraw-Hill Book Co. 1948. (*See* pp. 148–151.)

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Western Gall Rust (Woodgate Rust)

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Peridermium harknessii J. P. Moore, western gall rust, causes branch galls and trunk cankers of hard (2- and 3-needle) pines in North America. Trees of all ages are susceptible.

In spring, conspicuous pale-yellow blisters (aecia), occasionally preceded by droplets of clear, viscid liquid (pycnial exudate), emerge from the living bark of the globose rust galls and the marginal swellings of the trunk cankers. The aecia rupture a few days after their appearance and begin to release clouds of orange-yellow aeciospores. Carried by air currents, these spores may spread the disease for hundreds of miles. Dissemination of aeciospores and infection of young pine shoots continue for 1 to 3 months each year.

Each new infection is followed by the formation of a well-delimited gall sometimes accompanied by a small witches'-broom at the court of infection. The time it takes from when a rust gall develops until it produces its first crop of aeciospores varies between 2 and 4, and rarely 6, years. Thereafter the gall continues to enlarge, producing a new crop of spores each spring, until it has girdled the branch it grew on, resulting in the death of the branch, the gall, and the rust fungus that caused it. Rust galls cause growth losses and tree mortality but no cull, while the so-called "hip-cankers" of trunks cause severe cull but rarely mortality or even growth losses. The fungus may survive in cankers for as long as a century or two, causing conspicuous swelling and deformation of the bole but producing few if any aecia.

Peridermium harknessii may be recognized from the following characteristics: Pycnia, if present, and aecia on well-defined spherical to oblong, woody branch galls up to 8 cm. (rarely up to 30 cm.) in diameter, and on trunk cankers that commonly originate from branch galls adjacent to the trunk; small galls on 1- to 2-year-old twigs frequently taking the form of a small pear; aecia resembling irregular, yellow, and usually confluent blisters 1–8 mm. wide and 1–3 mm. high, rupturing laterally (circumscissile dehiscence), the peridial covers falling away in large flakes soon after dehiscence; filaments not abundant, projecting from the dome and the floor of the aecium, frequently continuous; aeciospores subglobose to obovoid and ellipsoid, 14–24 × 23–35μ; spore walls colorless and coarsely verrucose except for an elongate, lateral smooth spot.

Western gall rust is probably caused by a number of closely related species or races of fungi, some requiring alternate hosts for survival (see Hosts: Scrophulariaceae, below), some unable to infect alternate hosts and transmitting the disease by aeciospores from pine to pine, and some, as suggested by Meinecke, infecting both, alternate hosts and pine, with aeciospores ("facultative heteroecism"). Races not needing alternate hosts are a real danger to Old World hard pine forests.

The spread of western gall rust can be stopped by thorough inspection of susceptible pines in the field and the annual removal of *all* rust galls and cankers. The eradication of telial hosts (Scrophulariaceae, see below) in the vicinity of hard pines is of dubious value for rust control unless their susceptibility to the particular race of gall rust has been demonstrated. Systemic fungicides may become useful for chemical control of gall rust. Since symptoms of gall rust in pine may not appear for many years after infection has taken place, the only practical safeguard against accidental introduction and intercontinental spread of the disease is to prohibit entry of all living 2- and 3-needle pines from North and South America, except for experimental use following rigorous inspection and retention in quarantine for at least 4 years.

Range: In North America it occurs along the Pacific coast from Alaska to Mexico, eastward to the Atlantic coast from Nova Scotia to New York. Also found in Central and South America.

Hosts:

Pinaceae—

Indigenous in North America:

Pinus attenuata Lemm.
P. banksiana Lamb.
P. contorta Dougl.
P. coulteri D. Don
P. elliottii Engelm. var *elliottii* (as *P. caribaea* Morelet and *P. heterophylla* (Ell.) Sudw.)
P. engelmannii Carr. (as *P. apacheca* Lemm. and *P. mayriana* Sudw.)
P. jeffreyi Grev. & Balf.
P. muricata D. Don
P. ponderosa Laws. var *ponderosa*
P. ponderosa Laws. var *scopulorum* Engelm.
P. radiata D. Don.
P. sabiniana Dougl.

P. taeda L.
P. virginiana Mill.
Introduced to North America:
Pinus canariensis C. Sm.
P. densiflora Sieb. & Zucc.
P. halepensis Mill.
P. mugo Turra (as *P. montana* Mill.)
P. nigra Arnold var. *poiretiana* (Ant.) Aschers. & Graebn. (as *P. pinea* Habl.)
P. sylvestris L.
P. thunbergii Parl.

Scrophulariaceae—*Castilleja angustifolia* Nutt. var., *C. linariaefolia* Benth., and *C. miniata* Dougl. have been infected artificially with western gall rust; in addition, species of *Melampyrum*, *Orthocarpus*, *Pedicularis*, and *Rhinanthus* have been suggested as possible suscepts.

Literature:

Arthur, J. C. Manual of the rusts in United States and Canada. Purdue Res. Found., Lafayette, Ind., U.S.A. 1934.
Meinecke, E. P. Experiments with repeating pine rusts. Phytopathology 19: 327–342. 1929.
Peterson, R. S. Western gall rust on hard pines. U.S. Dept. Agr. Forest Serv., Forest Pest Leaflet 50. 1960.

Stalactiform Rust

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Peridermium stalactiforme Arthur & Kern, stalactiform rust, causes branch and trunk cankers of hard (2- and 3-needle) pines in North America. Trees of all ages, especially young trees, are susceptible.

In spring, droplets of clear, viscid exudate emerge from the pycnia on the living bark of the cankers, soon followed by conspicuous, pale-yellow blisters, the aecia of stalactiform rust. A few days later the aecia rupture, releasing clouds of orange-yellow aeciospores. The aeciospores infect alternate hosts (Scrophulariaceae, listed below) on the foliage of which uredinio-, telio-, and basidiospores are produced 3, 5, and 7 weeks after infection, respectively. The basidiospores are short-lived and cannot withstand extremes of temperature and moisture. Under suitable environment the basidiospores may infect susceptible pines growing nearby. The time between infection of pine, in late summer, and the appearance of the first crop of aeciospores on the young branches may vary from 10 months to 4 years depending on the age of the pines infected, climate, and other factors. There is no evidence that stalactiform rust spreads from pine to pine directly like western gall rust; host alternation appears to be obligate.

Peridermium stalactiforme may be recognized from the following characteristics:

Pycnia and aecia *on the roughened bark of elongate branch and trunk cankers* that reach a length of 30 feet or more but *do not*

cause any swelling of the wood, small cankers frequently diamond-shaped; *aecia resembling irregular, yellow, and usually confluent blisters* approximately 2 mm. wide and high, rupturing irregularly at the apex, the *peridial covers soon falling away* after dehiscence; filaments projecting from the dome and the floor of the aecium, *rarely continuous;* aeciospores subglobose to irregularly ellipsoid, 11–22 × 16–37μ, greatly varying in size and shape; spore walls colorless and moderately verrucose except for an elongate, lateral smooth spot.

Stalactiform rust may be mistaken for other stem rusts of pine that resemble it, particularly since aecia rarely develop on old cankers. Infected trees are frequently gnawed by rodents, causing severe resinosis and annual ridges at the margins of cankers.

Successful control depends largely on early recognition, particularly the recognition of large cankers on mature trees as being caused by this rust. As in white pine blister rust (*Cronartium ribicola* J. C. Fischer), long-distance spread can be avoided by repeated pruning of cankered branches and by prompt removal of trees with stem infections. New infection of pine can at least be minimized by the eradication of telial hosts in the immediate vicinity of susceptible pines. Systemic fungicides may be developed for efficient chemical control in the future. Since stalactiform rust may remain dormant and unrecognizable in pine for many years after infection, the only practical safeguard against accidental introduction and intercontinental spread of this dangerous disease is to prohibit entry of all living 2- and 3-needle pines from North America, except for experimental use following rigorous inspection and retention in quarantine for at least 4 years.

Range: In North America it is largely confined to the Pacific coast and Rocky Mountain regions from British Columbia to Idaho, but is found as far east as Manitoba and Minnesota, and believed to be transcontinental.

Hosts:

Pinaceae—
- *Pinus banksiana* Lamb.
- *P. contorta* Dougl.
- *P. jeffreyi* Grev. & Balf.
- *P. ponderosa* Laws.

Scrophulariaceae. Confirmed by infection with aeciospores.—
- *Castilleja angustifolia* Nutt.
- *C. applegatei* Fern.
- *C. coccinea* (L.) Spreng.
- *C. rhexifolia* Rydb.
- *Melampyrum lineare* Desr.

Probable additional suscepts.—Species of *Orthocarpus*, *Pedicularis*, and *Rhinanthus*.

Literature:

Arthur, J. C. Manual of the rusts in United States and Canada. Purdue Res. Found., Lafayette, Ind., U.S.A. 1934.

Mielke, J. L. The rust fungus (*Cronartium stalactiforme*) in lodgepole pine. Jour. Forestry 54: 518–521. 1956.

Wagener, W. W. Infection tests with two rusts of jeffrey pine. Plant Dis. Rptr. 42: 888–892. 1958.

Cedar Blight

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Phomopsis juniperovora Hahn, cedar blight, is a devastating disease of juvenile *Juniperus virginiana* L., *J. scopulorum* Sarg., and several other Cupressaceae species in the United States. Infected terminals and laterals of *J. virginiana* seedlings and transplants become light in color, then brown, and finally gray. Lesions on stems and branches frequently develop into cankers, which may result in girdling if stems and branches are of small diameter (one-half inch or less).

Black pycnidia develop on infected stems, branches, and leaves. Pycnidia are embedded at first, later becoming erumpent. Spores are extruded in tendrils. A-spores are hyaline, unicellular, ellipsoid, biguttulate, and commonly 7.5–10 × 2.2–2.8μ; B-spores are hyaline, unicellular, filamentous, slightly curved, and commonly 20.2–26.9 × 1μ. Intermediate type spores occur infrequently. A- and B-spores are produced in the same or different pycnidia. Perfect stage is unknown.

Infection is by A-spores, which presumably are distributed by water, wind, and insects. Infection in nursery stock occurs throughout the growing season. Infected seedlings and transplants (1 to 4 years old) of *Juniperus virginiana* and *J. scopulorum* are killed by girdling. Epidemics in the Great Plains have resulted in total losses in seedling and transplant beds of these two species. Pathogen has been isolated from 2- to 20-year-old natural reproduction of *J. virginiana*. Cankers seldom develop on such trees, thus mortality is negligible. Infected Cupressaceae species used as ornamentals appear unsightly because of numerous dead twigs; however, such trees are usually not killed.

Intercontinental spread could result from shipment of infected trees, seed lots containing plant debris, or logs and lumber with bark attached.

Range: In the United States it is common in the Great Plains and eastward to the Atlantic coast.

Hosts: Cupressaceae—

Chamaecyparis lawsoniana (A. Murr.) Parl.
C. obtusa (Sieb. & Zucc.) Endl. (Planted in U.S.)
C. pisifera (Sieb. & Zucc.) Endl. (Planted in U.S.)
Cupressus arizonica Greene
C. goveniana Gord.
C. lusitanica Mill.
C. macrocarpa Hartw.
C. sempervirens L. (Planted in U.S.)
Juniperus chinensis L. (Planted in U.S.)
J. communis L.
J. excelsa Bieb. (Planted in U.S.)
J. horizontalis Moench
J. lucayana Britt.
J. ashei Buchh.
J. pachyphloea Torr.
J. procumbens (Endl.) Sieb. & Zucc. (Planted in U.S.)
J. sabina L. (Planted in U.S.)

J. scopulorum Sarg.
J. squamata Lamb. (Planted in U.S.)
J. virginiana L.
Thuja occidentalis L.
T. plicata Donn
T. orientalis L. (Planted in U.S.)

Literature:

Hahn, Glenn Gardner. *Phomopsis juniperovora* and closely related strains on conifers. Phytopathology 16: 899–914. 1926.
——— Life-history studies of the species of *Phomopsis* occurring on conifers. Part 1. Trans. British Mycol. Soc. 15: 32–93. 1930.
——— Taxonomy, distribution and pathology of *Phomopsis occulta* and *P. juniverovora.* Mycologia 35: 112–129. 1943.

Phymatotrichum Root Rot

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Phymatotrichum omnivorum (Shear) Duggar—Phymatotrichum root rot, also known as Texas or cotton root rot—is a deadly disease of dicotyledonous plants. Monocots are immune. This root rot overwinters in the soil on live roots and by sclerotia. Growth through the soil is very limited; normal spread is by root contacts. Shear designated the perfect stage as a *Hydnum.* The mycelial stage is an *Ozonium* and its conidial stage *Phymatotrichum.* The *Ozonium* form is observed readily on infected roots as fuzzy, yellowish, acicular hyphae. P. B. Streets described and illustrated these various stages.

Following summer rains, the conidial stage may appear above ground in the form of spore mats. The spores are smooth, globose, averaging 4.8 to 5.5μ in diameter, or sometimes ovate with a measurement of 5 to 6 by 6 to 8μ. Spores do not germinate readily and fail to produce prolific hyphae. Spore mats, however, serve to designate the presence of this root rot. Besides wilted cotton or alfalfa and other agricultural crops, certain weeds such as ragweed and horsenettle serve as indicators of the presence of root rot. Diseased plants seldom give advance indications of infection, usually wilt suddenly and die. Root rot infected plants typically occur in roughly circular spots. Trees and shrubs at first may show a reduction in growth and vigor. Leaves of infected trees assume a yellow to bronze coloration and fall prematurely, or gradually drop off giving the crown a thin, sickly appearance. Resinosis is usually lacking.

Range: *Phymatotrichum omnivorum* is indigenous to the southwestern part of the United States and northern Mexico, extending from extreme eastern Texas into New Mexico, Arizona and southeastern California. It also has been found in Arkansas (SE. corner), Oklahoma (in the extreme south along the Red River), southeastern Nevada, and southwestern Utah. Reports of this root rot elsewhere in the world have not been verified.

Hosts: All dicotyledonous plants tested are susceptible in varying degree. Trees have been graded in susceptibility by Streets, Taubenhaus and Ezekiel, and more recently by Wright and Wells. A comparison of these ratings under different field conditions is given in Table 1.

TABLE 1.—*Rating of susceptibility of trees and shrubs to Phymatotrichum omnivorum root rot*

Hosts	Susceptibility rating by—		
	Streets	Taubenhaus and Ezekiel	Wright and Wells
Ailanthus altissima (Mill.) Swingle.	Very	High	Intermediate
Prunus armeniaca L.	do	do	Do.
Fraxinus velutina Torr.	Slight		
F. pennsylvanica Marsh.		Moderate	Intermediate
Catalpa speciosa Warder	Moderate	High	Do.
Juniperus virginiana L.	Slight	Slight	Resistant
Gymnocladus dioicus (L.) K. Koch.		Resistant	Susceptible
Populus deltoides Bartr.	Very	Extreme	Do.
Ulmus americana L.		High	Do.
U. crossifolia Nutt.		Resistant	Intermediate
U. pumila L.	Very	Extreme	Susceptible
Celtis occidentalis L.		Resistant	Resistant
Juniperus faccida Schlecht.		do	
J. scopulorum Sarg.		Slight	Resistant
Robinia pseudoacacia L.	Very	Extreme	Susceptible
Gleditsia triacanthos L.	do	do	Do.
Morus alba var. *tatarica*	Moderate	High	Intermediate
Eleagnus angustifolia L.	Slight	Resistant	Susceptible
Maclura pomifera (Raf.) Scheid.	do	do	Do.
Diospyros virginiana L.		Moderate	
Pinus nigra Arnold		do	Intermediate
P. taeda L.		Susceptible	Susceptible
P. ponderosa Laws.		do	Do.
Caragana arborescens	Slight	Slight	Slight
Sapindus drummondii Hook. & Arn.		Extreme	Resistant
Celtis laevigata Willd.		Resistant	
Platanus occidentalis L.	Slight	do	
Tamarix sp.	do	Moderate	Intermediate
Juglans major (Torr.) Heller	do		
J. nigra L.	Moderate	High	Intermediate
Chilopsis linearis (Cav.) Sweet	Resistant		Resistant

Control.—Losses from root rot in agricultural crops can be lessened to some extent by soil acidification, deep cultivation, crop rotations with monocots such as wheat, and addition of organic matter. For trees and shrubs in shelterbelts, no definite reduction is possible except perhaps by alternate planting of resistant with less resistant trees in a checkerboard pattern. Plants grown in root-rot areas should not be shipped elsewhere to similar climatic zones.

Literature:

Shear, C. L. The life history of the Texas root rot fungus, *Ozonium omnivorum* Shear. Jour. Agr. Res. 30: 475–477. 1925.

Streets, R. B. *Phymatotrichum* (Cotton or Texas) root rot in Arizona. Univ. Ariz. Tech. Bul. 71: 299–410. 1937.

Taubenhaus, J. J., and Ezekiel, W. N. A rating of plants with reference to their relative resistance or susceptibility to *Phymatotrichum* root rot. Tex. Agr. Expt. Sta. Bul. 527, 50 pp. 1936.

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Wright, E., and Wells, H. R. Tests on the adaptability of trees and shrubs to shelterbelt planting on certain *Phymatotrichum* root rot infested soils of Oklahoma and Texas. Jour. Forestry 46: 256–262. 1948.

Poria Root Rot of Douglas-Fir

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Poria weirii Murr. is a root rot of coniferous species common in Northwestern United States and southern British Columbia, Canada. The disease occurs in patches or centers of infection. Aboveground symptoms become apparent only after the disease has reached an advanced state. Shortening of the leader growth, sometimes accompanied by a distress crop of small cones on Douglas-fir, is followed by a thinning and yellowing or reddening of the foliage, and finally by death of the tree. Windthrow of living trees is common, the major decayed roots breaking close to the root collar.

Sporophores, forming on the underside of decayed logs and uprooted stumps, are found only periodically during the summer and early autumn. The resupinate fruit bodies are cinnamon buff to brown, usually with a broad to narrow white to cream sterile margin. On species other than western red cedar the sporophores usually form only a single tube layer; on cedar a perennial fruit body is formed. Setal hyphae and setae are abundant. Basidiospores are globose to subglobose becoming oblong-ellipsoid with a small apiculus, 4–4.9 × 2.8–3.2μ. No conidiospores are formed either in nature or in culture. Hyphae have simple septa, branching frequently immediately below the septum, but with no clamp connections.

The incipient stage of the decay is usually characterized by a crescent-shaped to spherical pattern of reddish-brown stain in the cross section of the stump. In the advanced state of decay, the wood breaks down to a yellow, laminated, pitted rot. Setae and setal hyphae are abundant in the laminated wood and a brown crustlike sheet often forms over mycelial masses on exposed wood surfaces.

This fungus, although capable of causing death to the majority of coniferous species in the Douglas-fir region, is most important as a root rot causing extensive losses in 25- to 125-year-old Douglas-fir and as a butt rot of cedar. The infection arises when roots contact the fungus present in roots and stumps of the previous crop. Spread to adjacent trees occurs when a healthy and diseased root are in contact. The role of basidiospores in the spread of infection is as yet unkown. It has been estimated that the productivity of second-growth Douglas-fir stands in western Washington and Oregon is, on the average, reduced by about 5 percent as a result of the activity of this fungus. A method for the economic control of Poria root rot is, as yet, unknown.

Intercontinental spread is possible through shipment of infected logs. Infection of seedlings below 6 years of age is not known.

Range: In North America it is common throughout the range of Douglas-fir in southern British Columbia, Canada, and northwestern United States. In Japan it is found in the subalpine forests of Honshu and the primeval forests of Middle Hokkaido.

Hosts: Pinaceae—

North America:

Pseudotsuga menziesii (Mirb.) Franco
Abies grandis Lindl.
Tsuga heterophylla (Rafn.) Sarg.
Pinus monticola Dougl.
Abies amabilis (Dougl.) Forbes
Abies lasiocarpa (Hook.) Nutt.
Pinus ponderosa Laws.
Picea sitchensis (Bong.) Carr.
Larix occidentalis Nutt.

Japan:

Tsuga diversifolia (Maxim.) Mast.
Abies mariesii Mast.
Abies sachalinensis Mast.
Picea jezoensis (Sieb. & Zucc.) Carr.

Cupressaceae—

Thuja plicata D. Don
Chamaecyparis sp.

Literature:

Aoshima, Kiyowo. Wood-rotting Poria from Japan II. Govt. Forest Expt. Sta. Bul. 59, Tokyo, Japan. 1953.

Buckland, D. C., Molnar, A. C., and Wallis, G. W. Yellow laminated root rot of Douglas-fir. Canad. Jour. Bot. 32: 69–81. 1954.

Childs, T. W. Laminated root rot of Douglas-fir. U.S.D.A., Forest Service, Forest Pest Leaflet 48. 1960.

Mounce, Irene, Bier, J. E., and Nobles, Mildred K. A root-rot of Douglas-fir caused by Poria weirii. Canad. Jour. Res. C, 18: 522–533. 1940.

Fir Tip Blight

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Rehmiellopsis balsameae Waterman is a blight of the current season's needles of several species of fir in Northeastern United States. The disease, which affects all sizes and ages of fir trees, appears first on the lower branches; usually the lateral twigs of these branches are more severely affected than the terminal twigs. Severe infection repeated for several years may result in the death of small trees; large trees are rarely killed. The disease causes relatively little damage to forest trees but may be very disfiguring and damaging to ornamentals.

Weather conditions exert an important influence on disease severity. A late, moist growing season leads to rapid development of young succulent tissue that is particularly susceptible to infection and

damage. An early growing season or one with limited rainfall results in less disease buildup.

The earliest symptoms appear on needles of the current season's growth when the bud scales begin to slough off. Yellowish-pink spots show on the needle tissue uncovered by the loosening bud scales. The young, developing twig, bearing these needles, usually continues to grow for a time, but before reaching maturity may turn dark brown or black and become shriveled, slightly curved, and brittle. Needles on such twigs change in color from light green to yellowish pink, then to dark reddish brown, and finally to gray. As these color changes occur, the needles dry out and their margins roll backward toward the lower surface, thus appearing narrower than healthy needles. They also frequently curve or bend. Adventitious buds may develop below the dead tips and produce weak, stunted needles late in the season. Infected needles are very brittle, but most of them overwinter on the twigs for one season and sometimes two.

The fungus may enter the twig tissue from an infected needle on *Abies concolor*, producing small cankers around leaf scars. Such cankers have not been observed on other species of fir.

About a month or 6 weeks after the first evidence of the disease, small, black fruiting bodies of the fungus appear in the tissue of the upper leaf surface. These develop slowly during the summer and reach maturity the following spring. The fruiting bodies also develop on shriveled twigs and on the small cankers at the base of infected needles. Ascospores are dispersed at the time new needles are being produced.

The fir tip blight in North America is very similar to the blight previously known in Europe. The causal organism is a distinct species from the European *Rehmiellopsis abietis* (E. Rostr.) O. Rostr. (Syn: *R. bohemica* Bub. & Kab.) with which it was once confused. The ascomata, asci, and ascospores of the American species are larger than the European. The ascomata of *R. balsameae* are 200–250μ in diameter. The asci are conspicuously thickened at the apex, being 5 to 8μ at the thickest point. The 16-spored asci are 81–141 × 33–41μ. The fusiform-elliptic ascospores are hyaline, 1-septate, cells unequal, sometimes slightly constricted at the septum, densely granular, straight or curved, and 31.5–49.9 × 6.3–12.6μ. A conidial stage has not been observed.

Hosts and Range: Pinaceae—

Abies concolor (Gord. & Glend.) Lindl. in Maine, New Hampshire, Massachusetts, Rhode Island, and New York.
A. balsamea (L.) Mill. New England States and New York.
A. cephalonica Loud. Rhode Island.
A. procera Rehd. Massachusetts.
A. fraseri (Pursh.) Poir. Massachusetts.

Literature:

Waterman, Alma M. New hosts and distribution of *Rehmiellopsis bohemica*. Phytopath. 27: 734–736. 1937.
———. Tip blight of species of Abies caused by a new species of *Rehmiellopsis*. Jour. Agr. Res. 70 (10): 315–337. 1945.
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———, and McKenzie, M. A. A disease of Colorado fir. Phytopathology 23: 108–109. 1933.

Brown-Spot Needle Blight

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Scirrhia acicola (Dearn.) Siggers is a needle spot and dieback mainly of *Pinus palustris* before height growth starts. The common spot is straw yellow at first, often with chestnut-brown borders which may become dark purplish in autumn; individual spots are about 3 mm. across but several often coalesce, and as the needles die from the tips, the green tissue between spots shrinks more, resulting in an embossed appearance. Some spots consist of an amber-yellow band with a small brown center.

Conidial stage is *Lecanosticta acicola* (Thum.) Syd. Acervuli innate-erumpent, at first globose, 50–100μ across, becoming elongate up to 1.5 mm. Conidia mostly 1–3 septate, sickle-shaped, 0.5–4 × 27–32μ, pale brown, discharged through clefts in epidermis in sticky matrix, disseminated mostly by rain splash.

Ascigerous stage: stromata compact, linear, non-clypeate, innate-erumpent, 1–18 locules mostly in single row, 40–80μ diameter, not distinctly ostiolate; asci aparaphysate, 6–9 × 30–35μ, 8-spored; spores hyaline, 1-septate, oblong-cuneate, 3–4 × 9–16μ. Ascigerous locules often develop marginally to acervuli. Conidia produced all year; ascospores mostly in winter and spring.

Brown spot is one of the major obstacles to the successful regeneration of *Pinus palustris* over wide areas in SE. United States. The disease causes chronic defoliation which either kills seedlings or delays initiation of height growth for many years. This pine is typically very resistant after reaching a height of 75 cm. *P. taeda* may be heavily attacked at all ages but most dieback occurs in the lower crown late in the growing season and, therefore, is less deleterious. Occasionally the fungus causes serious needle dying on other pines, including *P. strobus*.

Intercontinental spread through shipment of seedlings is highly probable, for even with rigid fungicidal spraying some infection occurs in most pine nurseries. Pollen and seed shipments appear safe.

Range: Common in the coastal States from North Carolina to Texas. Also reported inland as far as Missouri and Ohio. Occasionally in SW. and NW. United States.

Hosts: Pinaceae—

Pinus palustris Mill.
P. taeda L.
P. elliottii var. *elliottii* Little & Dorman
P. strobus L.
P. echinata Mill.
P. glabra Walt.
P. rigida Mill.
P. serotina Michx.
P. virginiana Mill.

Also occurs on following introductions to eastern United States:

P. attenuata Lemm.
P. contorta Dougl.

P. coulteri Don
P. halepensis Mill.
P. jeffreyi Grev. & Balf.
P. latifolia Sarg.
P. nigra poiretiana (Ant.) Aschers. & Graebn.
P. muricata Don
P. pinaster Ait.
P. pinea L.
P. ponderosa var. *scopulorum* Engelm.
P. radiata Don
P. sabiniana Dougl.
P. sylvestris L.
P. thunbergii Parl.

Literature:

Siggers, P. V. The brown spot needle blight of pine seedlings. U.S. Dept. Agr. Tech. Bul. 870: 1–36. 1944.

Wolf, F. A., and Barbour, W. J. Brown-spot needle disease of pine. Phytopathology 31: 61–74. 1941.

Septoria Canker of Poplars

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Septoria musiva Pk. (Perfect Stage—*Mycosphaerella populorum* G. E. Thompson) is a leaf disease and perennial stem canker of exotic and hybrid poplars in North and South America. The first leaf lesions appear from 3 to 4 weeks after the opening of the buds, mostly confined to leaves on the lower branches. Later the infection becomes general throughout the trees. The fungus produces necrotic spots of various shapes and sizes which often coalesce to involve large areas of the leaf. The individual lesions are brown with yellowish to white centers, and small black pycnidia develop throughout the lesions on both leaf surfaces. Under moist conditions the conidia are discharged from the pycnidia as curled, pinkish cirri. The conidia are hyaline, continuous to four (mostly two) septate, measuring from 17 to 57μ long.

The canker stage originates in the bark of twigs of the current year, entering the host through mechanical wounds, lenticels, stipules, or leaf petioles. By early summer a very conspicuous symptom is the presence of one or more dead leaves on the leaders, at the ring scars, or on the axillary branches produced on 2-year-old stems. At the bases of the dead leaves cankered bark is evident, which is usually black, frequently enclosing yellowish to white areas in which the pycnidia may be found.

The cankers may girdle and kill the leader and axillary branches during the first year and later spread from the axillary branches into the main stems. On entering the main stem the pathogen may produce a perennial canker resulting in considerable malformation of the main stem. Isolations from the diseased bark at the margin of older cankers frequently produce cultures of *Cytospora chrysosperma* (Pers.) Fr., and it is possible that advanced cankers may result from a combined attack of Septoria and other fungi such as *Cytospora*.

Perithecia of the *Mycosphaerella* stage occur on overwintered leaves and on the cankered bark of 1-year-old stems. The asci, which contain 8 spores, are cylindrical, short stipitate, from 51–73μ × 12–17μ. The ascospores are hyaline, one-septate, measuring from 17–24μ × 4–6μ.

This disease has caused serious losses on exotic and hybrid poplars in eastern and central North America. Although trees of all ages are susceptible, the canker stage is restricted to the bark on younger stems and branches.

Intercontinental spread is possible through shipment of infected trees or cuttings or of infected bark on logs or lumber. Importation of living plant material other than pollen or seed should be forbidden except for experimental use following rigorous inspection and retention in quarantine until all latent infections have had time to appear. Export logs and lumber should be debarked.

Range: In North America it is common in eastern and central Canada and the United States. In South America it occurs in Argentina.

Hosts: Salicaceae. Numerous exotic and hybrid poplars have been found to be susceptible particularly those with black, balsam, and cottonwood parentage.

Literature:

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Strumella Canker of Oaks

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Strumella coryneoidea Sacc. & Wint. (Imperfect stage). *Urnula craterium* (Schw.) Fries (Perfect stage). A perennial, usually nongirdling stem canker of *Quercus* in eastern North America. The first symptom is a yellowish bark discoloration centered around a dead branch or stub, usually with a raised margin and a depressed inner zone. On the tightly adhering bark near the center of the canker, small black nodules composed of interwoven hyphae are formed. They produce no spores. As the infected area enlarges, two types of lesions can result, target or diffuse, with frequent intergradations.

Target-type cankers are most common and result from the formation of successive ridges of callus in opposition to the slow growth of the fungus. These ridges form concentric circles around the base of the dead branch at the canker center. Usually there is a pronounced distortion or flattening of the cankered stem. The wood under the canker face slowly decays and eventually the bark sloughs off. Diffuse-type cankers occur when the fungus grows rapidly enough to girdle the stem before callus formation takes place, usually on stems that are under 4 inches in diameter. Water sprouts often appear be-

low the cankers. Small trees are killed but larger trees may remain alive for several decades or until the tree breaks at the canker.

The fungus rarely sporulates on living trees; however, if the tree dies, and while it is still standing, sporodochia are produced abundantly, both on the cankered area and beyond. They are dark brown, rounded, powdery pustules, 1–3 mm. in diameter, bearing stout branched conidiophores from the tips and sides of which are produced irregularly globose to pyriform, brown, spiny conidia, 6.7–8.1 × 4.7–5.8μ. These conidia do not germinate. They are windborne but their role in causing new infections is unknown.

After infected trees fall, the *Urnula* stage is produced. The apothecia, always attached to decaying wood, are common and conspicuous in early spring. They are up to 3–4 cm. in diameter and 4–6 cm. deep, black, leathery, rupturing irregularly. The asci are cylindrical, with 8 one-celled, smooth, hyaline ascospores, 12–14 × 25–35μ. Ascospores are windborne and germinate readily within a few hours after release from apothecia.

Strumella canker is common but losses are not catastrophic. The fungus spreads slowly, seldom infecting more than 2 or 3 percent of the trees in an oak stand. Most cankers, however, occur on the first 12 feet of the trunk, seriously reducing or destroying the value of the butt log. Wind breakage of cankered trees occurs frequently. Control is through sanitation—removing infected trees in weeding, thinnings, and harvest cuts.

Prevention of intercontinental spread of Strumella canker should not be difficult. Infected trees are distorted and the infected portion is seldom, if ever, harvested and utilized for anything other than fuel.

Range:

In North America it is common in eastern United States. The fungus is reported as a saprophyte in eastern Canada, western United States, and one collection from Europe (Germany). Important only on oaks.

Hosts:

Fagaceae—
Quercus velutina Lam.
Q. rubra L.
Q. coccinea Muenchh.
Q. ilicifolia Wangenh.
Q. macrocarpa Michx.
Q. marylandica Muenchh.
Q. prinus L.
Q. prinoides Willd.
Q. palustris Muenchh.
Q. bicolor Willd.
Q. alba L.
Fagus grandifolia Ehrh.
Castanea dentata (Marsh.) Borkh.
Betulaceae—*Ostrya virginiana* (Mill.) K. Koch
Tiliaceae—*Tilia americana* L.
Cornaceae—*Nyssa sylvatica* Marsh.
Aceraceae—*Acer rubrum* L.
Juglandaceae—
Carya glabra (Mill.) Sweet
C. ovata (Mill.) K. Koch

Literature:

Davidson, R. W. *Urnula craterium* is possibly the perfect stage of *Strumella coryneoidea*. Mycologia 42: 735–742. 1950.

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Dwarfmistletoes of Conifers

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Arceuthobium spp. are conifer-inhabiting flowering plants of the mistletoe family (Loranthaceae). Leaves are reduced to scales, opposite, decussate, inconspicuous, same olive-green color as the stem. The internodes are round or rectangular in cross section, depending on species and age.

The larger species (*A. campylopodum* and *A. vaginatum*) are commonly up to 10 cm. long, while the smaller ones (*A. douglassi* and *A. pusillum*) are usually no more than 1.5 cm. The plants are unisexual. Staminate flowers are about 2–4 mm. in size, 3- or 4-partite, deciduous, anthers sessile with ringlike archesporium around a central columella, pollen tricolpate, spinulose. Pistillate flowers are about 1 mm. in size, little differentiated, with two perianth segments greatly reduced, liplike around the short and blunt stigma. Fruit at maturity is a recurved, turgid berry, ovate to elliptical in shape, with a conspicuous transverse color demarcation.

The fruit is explosive, releasing the single seed upwards or obliquely away for many feet. The seed, which consists of a single cylindrical and largely undifferentiated embryo surrounded by a large amount of chlorophyllaceous endosperm, germinates on the surface to which it has become attached. If this surface is a young branch of a suitable host, the radicle penetrates into living tissue and a new individual develops. Since the remainder of the seed dies or falls away, dwarfmistletoes have a completely internal stage which may last for more than a year. Flowering time varies for the different species, but expulsion of the seed always takes place in the fall.

The haustorial or endophytic system is a complex, ramifying system divisible into a longitudinal system of strands, external to and more or less parallel to the host cambium, and a radial system consisting of sinkers radially oriented in phloem and xylem, such sinkers taking their departure from cortical strands. Direct xylem-to-xylem connections are common between the largely vascular older sinkers and the host tracheids. Cortical strands and perhaps all sinkers originate as uniseriate filaments of cells each with a single apical cell, but divisions in the more distal cells turn the strands into vasculated structures much like normal roots.

Symptoms of the disease vary not only between species of dwarfmistletoe, but also within one species when several host species are involved. The simplest response of the host is a localized, somewhat fusiform swelling. In this case the endophytic system is confined to

the swelling, the youngest tips growing outward near the poles of the spindle. Flowering shoots first appear near the original court of entrance, but succeeding ones emerge in concentric zones of increasing diameter. The center of such an infection usually deteriorates and forms a court of entrance for various decay-producing fungi and bark beetles.

Some dwarfmistletoes, but only on certain host species, call forth the production of a witches'-broom in which suppression of lateral branches is largely released, resulting in a dense growth of abnormal appearance. In many of such brooms the endophytic system of the dwarfmistletoe pervades all branches, even to penetration of the apical meristems of the host, and these mistletoe shoots are present in predictable patterns along these host branches. In most instances the uninfected part of the tree deteriorates badly, leaving only the broomed portion alive.

Since the susceptibility of many exotic conifers has been demonstrated and some native hosts are planted extensively abroad, the North American species of *Arceuthobium* pose a considerable threat. Fortunately, the seeds are not long-lived and lose their ability to adhere to new objects after some time, therefore the chance of establishment on other continents by means of seed is small. A greater danger, perhaps, lies in the export of trees of nursery size, since these may bear invisible infections. Experience in North America has shown complete elimination of an infected stand to be the only possibility of eradication.

Dwarfmistletoes are difficult to evaluate as to their economic importance. There is general agreement, however, on the severity of losses in certain areas. Field observations point to significant losses in the following instances:

Hosts: Pinaceae	*Dwarfmistletoes*	*Area*
Abies spp	*Arceuthobium campylopodum.*	Sierra Nevada, California.
Picea spp	*A. pusillum*	Northeastern United States and across Canada to Manitoba.
Pinus spp	*A. campylopodum*	Eastern Oregon south through most Pacific and Southwestern States.
P. banksiana, P. contorta.	*A. americanum*	Northern Alberta to Colorado.
Pseudotsuga menziesii	*A. douglasii*	Interior southern British Columbia to northern California and the Southwestern States.
Tsuga heterophylla	*A. campylopodum*	Pacific Coast, Alaska to Oregon.

A more complete listing of hosts is provided in Kuijt (1955).

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Principles of Forest Disease Control

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I feel presumptuous in discussing the principles of forest disease control before this group since most of you are specialists in the field, and therefore anything I can say will sound trite. However, there will probably be some disagreement with my ideas, and reasoned disagreement is invaluable for the complete exploration of any topic.

There is often such a difference in the effect of diseases caused by native pathogens on forests that control measures differ widely. For native diseases these measures are largely indirect—that is, applied as a part of routine silvicultural practice; whereas, for introduced diseases, direct measures must often be used—that is, special operations solely against the disease. Whenever possible, indirect measures should be employed. Diseases caused by native pathogens do not threaten the commercial extinction of a native tree species, but diseases caused by introduced fungi may eliminate, and in certain cases have eliminated, important timber species. Consequently, there is usually far greater urgency to control diseases caused by introduced pathogens than to check those caused by native pathogens.

Control measures are limited since forest stands in general have a relatively low value considering the years required to produce them, therefore expenditures for disease control must be modest. Thus, the intensive methods of disease control that are routine in agriculture and horticulture are possible only occasionally in forestry—the exception being nurseries where intensive control measures are standard practice. Insofar as possible, control should be part of regular silvicultural practice.

Certain precepts should be pursued in order to minimize disease. Each of us can cite exceptions to these precepts and, because of conditions in some forest regions, it may seem necessary to disregard them. When this is done, it should be with realization of increased expenditures that may have to be made for disease control. Some of these precepts follow:

Seedling stands are better than sprout stands. Natural regeneration is better than artificial, particularly planting. Uneven-aged stands are better than even-aged. The selection system is better than clear cutting. Mixed stands are preferable to pure stands, and mixtures of conifers and hardwoods are especially desirable. Natural stand composition should be followed as far as possible. Of course in certain forests nature's method is pure stands which should be accepted. Good sites are essential—no amount of care can produce a satisfactory stand on an unsatisfactory site. Native species are safer than exotics, because exotics are more liable to injury when conditions become critical. Because of the high risk of bringing in new pathogens, exotics should be introduced as seed only. The seed must be of proper origin both as to location and character of the mother trees.

Our safeguard against introduced pathogens is quarantines which admittedly are measures of delay and not measures of exclusion.

Nevertheless, delay is more important in forestry than in agriculture because of the long time necessary to produce a crop of trees and to work out control methods when disease does appear.

The control or extermination of a pathogen by another pathogen—that is, biological control—theoretically should be the cheapest and most effective method of dealing with pathogenic diseases. Yet so far there has been no instance of any worthwhile success in this field against fungi and related organisms, and nothing now appears promising for the future.

The development of resistant trees by selection or hybridization, when this can be accomplished, is probably the best method for controlling disease. The limitations of the method are the necessity for developing several resistant hybrids or selections and the long time this requires. This means that in dealing with a disease caused by a virulent pathogen a successful method of control must be developed quickly so the affected tree can be perpetuated until such time as sufficient resistant individuals can be obtained.

Finally a new and promising method of control is being developed; namely, the use of systemic fungicides. Lesions of white pine blister rust on western white pine trees have been reported to be inactivated within 18 to 24 months after the boles of the trees have been sprayed to saturation from ground level to a height of 5 feet with 150 parts per million of cycloheximide in fuel oil. This mixture also shows promise as a foliage spray against blister rust. The use of systemics may bring revolutionary changes in control methods in the next decade or so.

LESSENING THE THREAT OF INTERNATIONALLY DANGEROUS TREE DISEASES

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The maintenance of sustained yields in high quality timber is one of the prime aims of research in forestry. Among the enemies of sustained yields are the following: fire, insects, animals, inroads of "civilization," and diseases. In the disease category one finds not only the pathogenic agents that destroy foliage and kill trees, but also those that cause root, butt, and trunk rot. In the United States, the total growth impact of forest diseases is estimated to be about 45 percent of the losses caused by all destructive agents.

In North America the invasion of foreign forest tree diseases has caused tremendous losses. Examples are chestnut blight, white pine blister rust, and Dutch elm disease. The latter is insect borne. The importance of injurious insects that might come from abroad also is clearly recognized. However, the insects are outside the scope of the present consideration.

Epidemic diseases are much more serious for forest trees than for comparable agricultural crops. If a field of wheat should be destroyed, something else could be planted the next year. But, if the trees are damaged, then 10, 25, or 50 years of the productivity of the land may be reduced or lost. Furthermore, with some of the root and butt rot organisms another danger appears. Such pathogens may develop in an apparently innocuous way in the nurseries. Thus, dangerous disease organisms may be distributed with the nursery stock used for reforestation throughout the areas.

A program has been initiated to investigate (1) what the chances are that certain internationally dangerous diseases might be moved from one country to another, (2) how this movement could be prevented, or at least slowed down, (3) how quarantines could be made more effective, (4) how eradication might be accomplished if a foreign disease should appear, (5) how its damaging effects might be avoided or reduced if it appeared and could not be eradicated, and (6) how research might supply badly needed information about the host, the pathogen, and disease development. To facilitate such a program, the writer visited sixteen different countries to consult with leading foresters, quarantine officials, forest geneticists, and pathologists. His trip was financed by the University of Wisconsin.

Numerous suggestions on what to do have come from many competent people. A summary appears below:

Directory of Critical Tree Diseases.—In order to have available information about the epidemic diseases and their importance in each country, annotated lists of them have been, or are being, prepared in

a number of places. From such lists, distribution maps can be made. At the same time, the maps may show the distribution of reporting pathologists rather than that of the disease. Negative reports may be significant only if a qualified person has made a diligent search.

Such lists may not disclose diseases which appear innocuous in one country but which might be dangerous in others (such as the chestnut blight in east Asia). However, research, as discussed later, may bring these to light. An example of such an annotated list is given for the United States by Spaulding in USDA Agricultural Handbook No. 139 where 33 diseases come under the heading "American Forest Diseases Potentially Dangerous to Forests of Foreign Countries."

Much can be learned by a study of surviving exotic trees in plantations already made, as Spaulding has done.

List of Key Men in Each Country.—A list is being prepared of one or more men from each country active in forest pathology. These men will exchange critical information, arrange for research on an international basis between suitable individuals, and advise in case of an outbreak.

Strengthening Quarantines.—Plant quarantines are the accepted bulwark against the entry of dangerous tree diseases into a country. Modern air travel greatly increases the chances for distributing pathogens from one country to another. Many foresters are concerned about the effectiveness of quarantines because, no matter how well they are administered, dangerous pathogens may get through. Forest pathologists have an obligation to supply to quarantine officials a list of the most dangerous pathogens and to describe the diagnostic symptoms by which they may be recognized. At the same time interferences with commerce, travel, and the exchange of scientific materials need to be held to a minimum compatible with protection. More research is needed if quarantines are to achieve maximum dependability.

Organizations Now Operating.—Various organizations deal in one way or another with certain aspects of these foreign tree disease problems. The names of some important ones follow: European and Mediterranean Plant Protection Organization; Moscow-Peking Convention; Plant Protection Committee for the South East Asia and Pacific Region; Inter-Africa Phytosanitary Commission; Organismo International Regional de Sanidad Agropecuaria (Central America); Plant Quarantine Division, ARS, U.S. Department of Agriculture; and Division of Plant Protection, Canada Department of Agriculture. In addition, the Food and Agriculture Organization of the United Nations in Rome is interested in reducing the spread of forest pathogens from one continent to another and can supply the name and address of the quarantine services in all member countries.

Cooperative International Research.—For a number of critical problems no one knows the answers. To secure them, cooperative research is essential between men in different countries. Obviously, one cannot move either the pathogens or the living trees that might carry them from one country to another except with extensive precautions. However, disease-free selected or treated seed can go from one country to a suitable place in another country with relatively little danger.

Some important topics for research follow:

(a) *Host ranges.* The different kinds of trees attacked by critical pathogens need exploration. In some cases related ornamentals carry

tree diseases. Such research may best be done in nature at a location where the pathogen is active.

(b) *Alternate hosts.* Especially with the rusts (e.g., white pine blister rust has one critical stage on currants and gooseberries) the various plants necessary for the development of certain stages of the fungus need to be characterized with precision.

(c) *Environmental influences.* Temperature, moisture, light, and mineral nutrition have important influences favoring or discouraging epidemics. The study of microclimate is essential for knowing how the pathogens work and how their damage may be prevented.

(d) *Disseminating agents.* The potentialities of different carriers of pathogens need investigation, especially insect vectors.

(e) *Seemingly innocuous pathogens.* Attempts should be made to disclose dangerous pathogens that are seemingly unimportant in their native country. Chestnut blight was considered of no consequence in east Asia, but when the pathogen reached America, it practically eliminated the American chestnuts.

Suitable experimental plantings might disclose such wolves-in-sheep's-clothing. (The technique for such research might follow the work done with the world collection of wheat).

(f) *Improved methods for eradication.* If a dangerous disease should appear in a limited area of a country, eradication might be tried. Fire and chemicals have been useful, but certain other procedures also may be valuable. To be most effective, a technique for eradication should be developed in the country where the disease is active. The results should be available for immediate use to countries where the disease might appear as a new invader.

(g) *Disease resistance.* In many countries native trees fail to meet the needs for reforestation or afforestation. Trees from other countries have been widely and advantageously used, but sometimes with disastrous results from disease. Furthermore, tree breeders are continually developing improved trees. In both cases tests for resistance to dangerous diseases are essential. The selection and development of disease resistant trees hold great promise.

For many kinds of research involving pathogens and isolation, several locations are needed. An island with a mountain might provide many desirable conditions. Changes in moisture occur from the rainy to the dry side. Changes in temperature appear at higher or lower elevations. In some mountain valleys the length-of-day would be shortened. In such isolated places pathogens might be studied effectively with little danger of spreading the diseases.

Relations with Agricultural Research Stations.—In many countries the forest pathologist works in relative isolation from men trained and experienced in handling various field, forage, fruit, fiber, and plantation-crop diseases. Such men usually have a background of information and "know-how" as well as equipment that the forest pathologist could use with advantage. For the most part, all this would be available merely for the asking. Active cooperation between forest pathologists and agricultural pathologists deserves not only encouragement but also the active removal of any physical and administrative barriers that prevent their working together.

In conclusion, the possibilities are dismaying that internationally dangerous tree diseases may move from one country to another and upset sustained yields. However, international understanding, co-

operation, and research can do much to slow down or even to prevent more catastrophes like that caused by chestnut blight in America.

Summary

1. *A directory of critical diseases* in each country is needed. For example, in the United States Spaulding lists 33 diseases potentially dangerous for other countries (USDA Handbook No. 139).
2. *A list of key men* is essential. At least one man per country should assemble and distribute information.
3. *Strengthening quarantines* is imperative, but interference with commerce should be minimal.
4. *Existing organizations* are adequate.
5. *International researches* essential for effective operations include: (a) improved methods for diagnosing diseases and the preparation of authoritative host ranges of dangerous fungi; (b) understanding of alternate hosts of rusts; (c) the critical importance of environment (e.g., temperature, moisture, light, and mineral nutrition), of seasonal development, and of microclimate to epidemic outbreaks; (d) the characterization of agents disseminating pathogens, especially insects; (e) the unmasking of seemingly innocuous pathogens (e.g., chestnut blight) by suitable foreign plantings; (f) improved eradication procedures to be developed where the diseases are rampant; and (g) the development of elite and resistant varieties.
6. *Improved cooperation* between agricultural experiment stations and forestry research stations would be mutually beneficial.

DISCUSSION AND RECOMMENDATIONS

There was general concurrence with Professor Riker's appraisal of the need for international cooperation to lessen the intercontinental spread of forest pathogens and with his suggestions for future actions to that end.

The Working Group agreed unanimously to this plan of action:

1. To stimulate the preparation of annotated lists of dangerous forest diseases in all continents.
2. To draft a roster of leading pathologists in all continents to whom may be sent inquiries about local forest diseases and information on threatening diseases.
3. To advise existing plant quarantine organizations of the potential hazard of forest diseases in other countries and continents.
4. To emphasize the need for increased research on forest disease prevention and control in all continents.

The Working Group approved a proposal that an international symposium should be organized to consider the particularly dangerous forest diseases of all continents and to recommend measures to lessen or prevent their intercontinental spread. It was also recommended that the Union should be requested to ask FAO to sponsor such a symposium at an appropriate time and place. The Working Group will assume all responsibilities for arranging for the technical subject matter coverage at the symposium.

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